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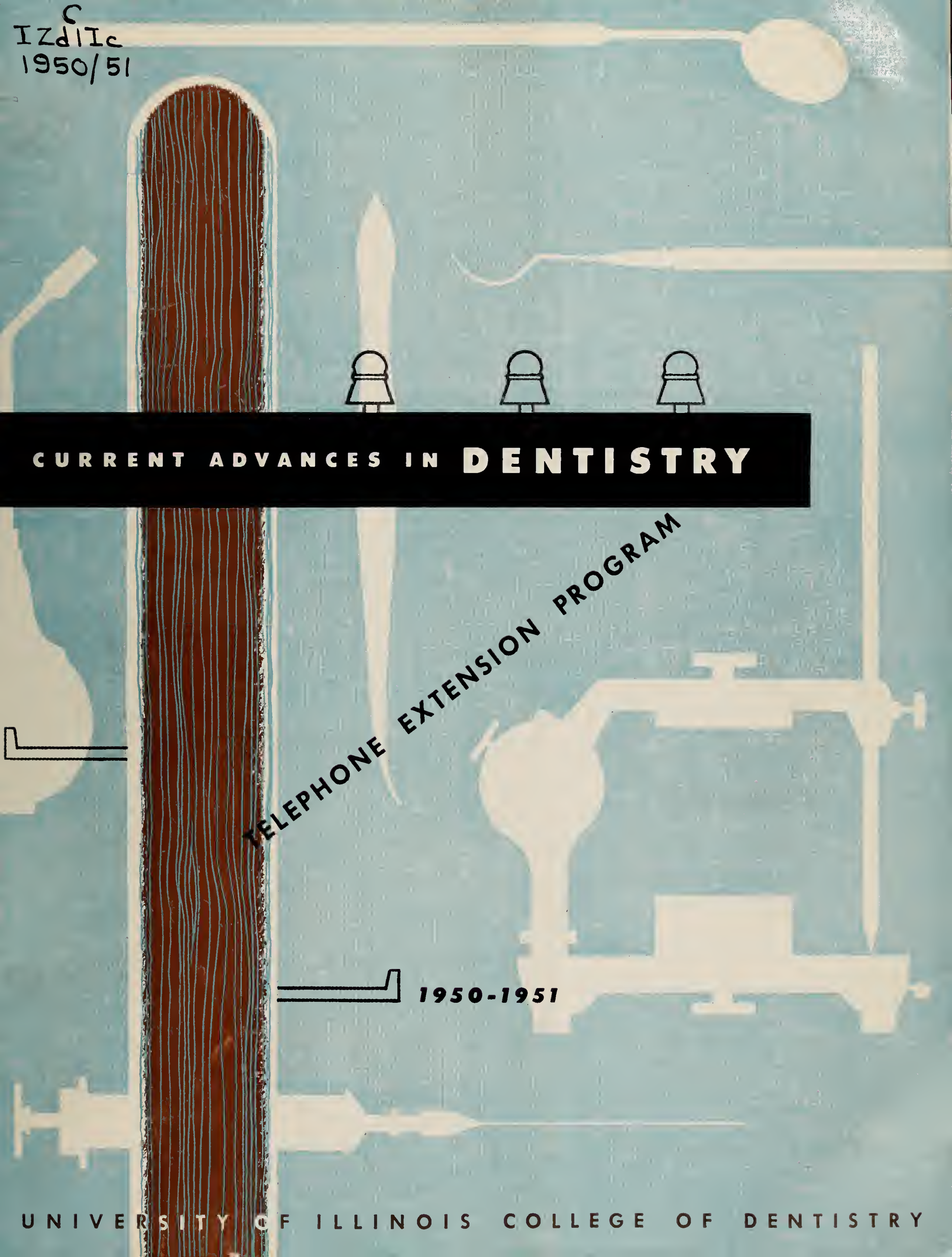

# CURRENT ADVANCES IN DENTISTRY

## TELEPHONE EXTENSION PROGRAM

1950-1951

UNIVERSITY OF ILLINOIS COLLEGE OF DENTISTRY

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# CURRENT ADVANCES IN **DENTISTRY**

*1950-1951*

PUBLISHED 1950 BY

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# CURRENT ADVANCES IN DENTISTRY

## 1950-1951

A MANUAL OF THE
UNIVERSITY OF ILLINOIS
COLLEGE OF DENTISTRY
TELEPHONE EXTENSION
PROGRAM

edited by
**ISAAC SCHOUR**
**and SAUL LEVY**

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The illustrations in this manual do not necessarily refer to the text. Some of the illustrations are planned as a part of the discussion only.

## TIME OF LECTURES

5:30 P.M. Pacific Coast Standard Time
6:30 P.M. Rocky Mt. Standard Time
7:30 P.M. Central Standard Time
8:30 P.M. Eastern Standard Time

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1950/51

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UNIVERSITY OF ILLINOIS
COLLEGE OF DENTISTRY
*Chicago, Illinois*

# FOREWORD

It is progress that creates the need for further progress. The telephone extension program was inaugurated with the idea of giving dentists in districts far distant from metropolitan areas the opportunity to keep up on current advances in dentistry. Machinery was set in motion to use the revolutionary experimental new technic of transmitting a series of symposia over the long distance telephone lines.

It was then discovered that dentists are not only desirous and anxious to keep abreast of the latest advances in dentistry, but are also interested in furthering their knowledge in the basic sciences. The first series of symposia was fairly well saturated with basic scientific teaching, yet was subscribed to by 6650 dentists from 163 cities in the United States and Canada. The total enrollment for the present series is not yet known. However, it will greatly surpass that number because there are at this writing 250 cities registered in the hookup, including practically all of the major metropolitan areas.

Postgraduate dental education has made rapid progress. It is hoped the telephone extension project will create the need for further progress and open new vistas for its successful fulfillment.

The manual is an integral part of this new system of long distance postgraduate teaching. This is the second volume of material prepared to accompany the panels arranged for telephone transmission by the Division of Postgraduate Studies of the College of Dentistry, University of Illinois. Like that of the first program on Current Advances in Dentistry given last year, it contains in abbreviated form the material that will be covered by the various speakers, together with the illustrations to which they will refer.

The enthusiastic approval given the manual last year was convincing evidence that it is an effective medium of instruction, and as a result several innovations have been introduced to improve it further. In those illustrations where it has been found particularly important, color plates have been added. Also, certain material which lent itself to such treatment has been set up in the form of supplementary charts which may be used if desired for ready reference at the operating chair.

The work of arranging, editing and publishing this manual is the greatest single task of the staff, and the second most expensive item in the telephone extension program. The close cooperation and enthusiastic support of a number of agencies which combined to make the extension program a success last year have been assured us again. The designing and publishing of the manual has been done by the Department of Mass Communications of the University; the generous support of the Kellogg Foundation has made possible certain experimental phases which could not otherwise have been undertaken, and the University radio station, WILL, will continue to assure excellence of transmission from Chicago. Last, but most important, has been the response of the profession, whose approval has been demonstrated by innumerable able letters of encouragement and by a great increase in enrollment over last year. To all of these we extend our heartiest thanks.

Allan G. Brodie

Dean

# HISTORY

The University of Illinois College of Dentistry Telephone Extension Program is the most unusual educational project ever attempted in the history of education.

***Inspiration 1947***

It was inspired by a postgraduate course in "Caries Control" offered in October, 1947. It was conceived by an alumnus hundreds of miles away. It was executed from the Illinois classroom to a meeting room in Scranton, Pennsylvania, 800 miles away. For the first time a course of study was made available over the long distance telephone wire to twenty dentists.

***1948***

In 1948, via the same method, courses were given to dentists in Scranton, Pennsylvania, New Iberia, Louisiana, and to six cities in the State of Illinois.

These experiments proved successful, and Dean Allan G. Brodie proposed to Dr. George D. Stoddard, President of the University, that this means of teaching should be made available to all dentists, regardless of location, who are eager for new information in the dental sciences. The proposal was approved and plans were then made to transmit a nation-wide program of round table symposia and discussions to interested groups of dentists.

***Present program begun 1949***

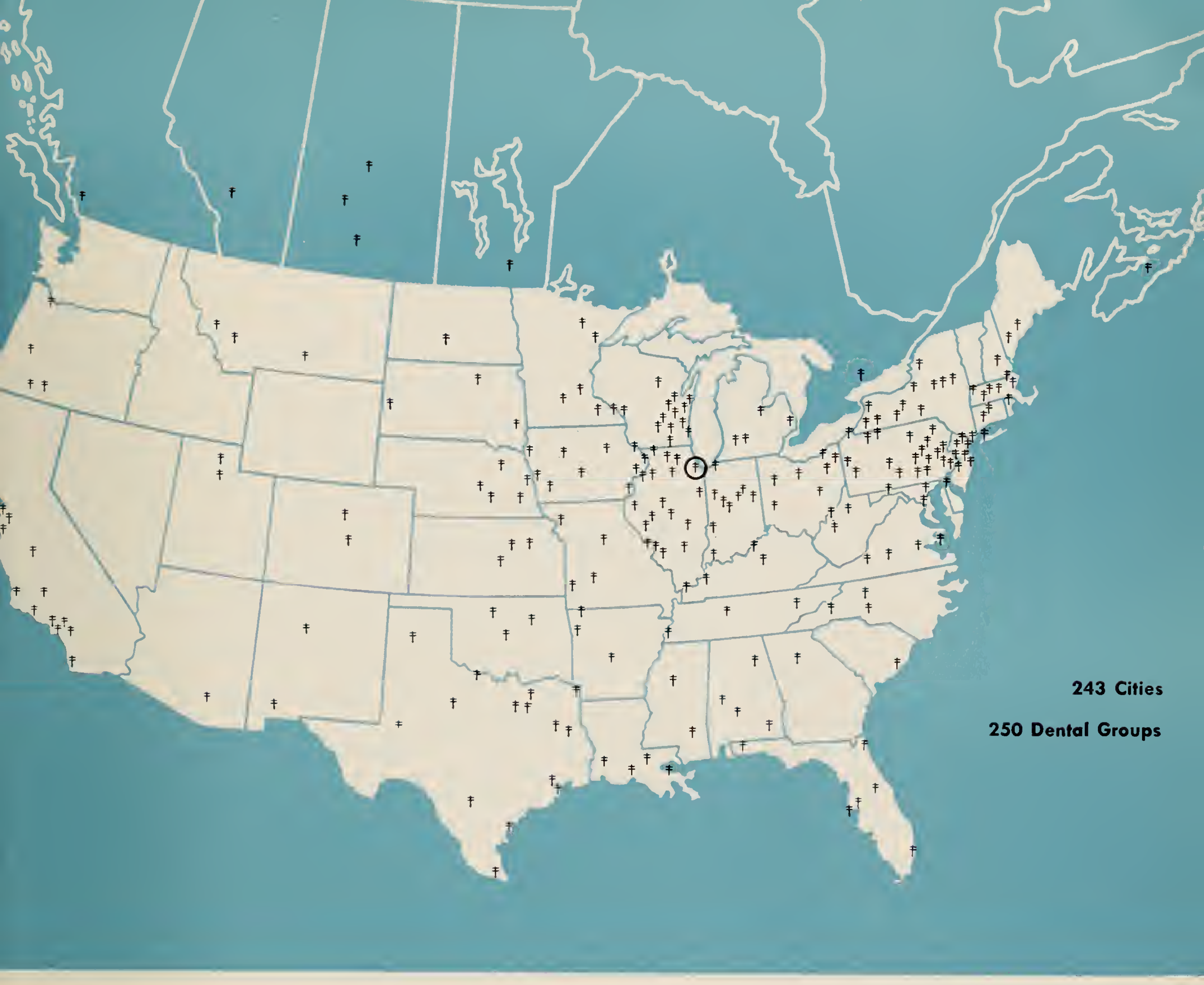
On October 10, 1949, just two years following the original experiment, the University of Illinois College of Dentistry transmitted by telephone the first of a series of round table symposia on six different phases of dentistry simultaneously to 138 dental groups from cities all over the United States and including Canada. On April 10, 1950, the final lecture of the Series was sent to 163 dental groups, representing a listening audience of 7,000 dentists from 42 states, the District of Columbia, and Canada.

***1950-1951***

This revolutionary approach to keeping the busy professional man up to date with scientific progress has been accepted with great acclaim. There are 250 dental groups registered for the present series of transmissions. Inquiries which have been received from Australia, Mexico, and South America, are further evidence of the wide popularity of this plan.

The mechanics are quite simple. They involve attaching microphones to the telephone circuit at the delivery end, and a loud speaker to the telephone circuit at the receiving end. However, the engineering details are many and complex. The labyrinth of over 18,000 miles of telephone wire must be carefully prepared and operated. It is important that the quality of the reception be clear, that there be no interference and no interruptions. The American Telephone and Telegraph Company assumed all responsibility for the technical details. They are doing an excellent job of obtaining splendid results. Since its inauguration even natural hazards, like storms and collisions, were unable to keep the enrolled groups from listening in.

There are three very interesting features connected with this program. First, it is possible for the dentists to receive first hand information about



the latest developments in dentistry. Second, this information is obtained from prominent educators and research workers from many universities in all parts of the country and in Canada. Third, regardless of residence each individual taking the course pays the nominal tuition fee of ten dollars for the entire series of lectures.

The success of the present vast network is due to the farsightedness of the many leaders from organized dentistry all over the country. It was because their imaginations were fired by the educational experiment that they worked with a tremendous drive and enthusiasm to give this newest form of education a chance to succeed.

This new technique for disseminating education, which was born two years ago, has already proved its right to a permanent place in the field of teaching.

SAUL LEVY, B.S., D.D.S.

Director, University of Illinois
Telephone Extension Program

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HONORS: Sigma Xi, Omicron Kappa Upsilon, Howard Taylor Ricketts Award, John R. Callahan Award.

# MODERN CONCEPTS OF DRUG THERAPY IN DENTAL PRACTICE

**Monday, November 13, 1950**

## OUTLINE

| | | |
|---|---|---|
| 1. | Changing Concepts of Drug Therapy | J. R. Blayney<br>Moderator |
| 2. | Rationale of Drug Therapy | T. J. Hill |
| 3. | Control of Pain: Analgesics, Sedatives, Narcotics | F. D. Ostrander |
| 4. | Control of Infection: Antibiotics and Sulfonamides | C. P. Miller |
| 5. | Control of Bleeding: Hemostatics | J. G. Allen |
| 6. | Hazards of Irradiation: Therapy on Oral Tissues | K. H. Thoma |

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Miller, C. P., M. Bohnhoff, 1950. The Development of Bacterial Resistance to Chemotherapeutic Agents. *Annual Review of Microbiology, Volume IV.*

Thoma, K. H., 1944. Oral Pathology, 2nd Ed., C. V. Mosby Co., St. Louis, Mo.

## SUMMARY

**CHANGING CONCEPTS OF DRUG THERAPY.** In previous decades dental therapy consisted primarily of manual technics for the removal of teeth, cavity and crown preparations for the reception of restorations, and the removal of calculous deposits from tooth surfaces.

Near the close of the first half of the Nineteenth Century, Wells and Morton demonstrated the anesthetic properties of nitrous oxide and ether. Approximately 30 years later Koller, an ophthalmologist, showed the effectiveness of cocaine as a local anesthetic. All of this led to the use of drugs for the relief of suffering during surgical procedures. Oliver Wendell Holmes beautifully described the state of anesthesia: "The fierce extremity of suffering was steeped in the waters of forgetfulness, and the deepest furrow in the knotted brow of agony was smoothed forever."

Because of the very nature of dental treatment the dentists have played an active role in the development of modern technics for the use of local anesthesia. Antiseptics, germicides, and astringents were other types of drugs which former practitioners of dentistry employed extensively. These were used without any consideration as to the character of the microorganisms which were invading the tissue. These drugs were for the control of infections within the dental pulp and periapical regions, as well as the gingival tissue.

Today the use of drugs in the dental office has changed materially. Basic research in the areas of physiology, pharmacology, bacteriology, physics, and hematology has provided us with a better understanding of oral disease and oral manifestations of systemic disease. It has provided also new preparations for the treatment of these conditions. Present day therapy is based on an understanding of the reactions produced by the drug employed. Today a direct and single medicament replaces the multiplicity of drugs formerly employed in therapeutic preparations.

Like therapy in all other fields these changes have taken place slowly, almost imperceptibly. The fact remains that today the dentist has many useful drugs for the favorable modification of the course of oral diseases. Whenever drug therapy is employed, it is well to remember the old axiom "Drugs sometimes cure, often relieve, but always console."

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AUTHOR: Oral Pathology.

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HONORS: Sigma Xi and Omicron Kappa Upsilon.
Chairman, Council of Dental Therapeutics, Revision Committee of United States Pharmacopoeia.

## RATIONALE OF DRUG THERAPY

Therapeutics is the treatment of disease. It applies to physical, thermal and mechanical measures as well as to the use of drugs, that is, chemical agents. Many of the therapeutic methods used in dentistry are of a mechanical nature. As the systemic relationship to oral disease is more fully understood and appreciated, the application of drug therapy is rapidly replacing some of the mechanical methods of therapy formerly employed.

Rational therapeutics is the application of therapeutic measures based upon a known pharmacologic action or on established physiologic response. When methods of treatment are without a sound basis for the therapeutic measures employed, they are irrational. When newer and more specific drugs are developed, former methods, rational or otherwise, become obsolete.

Empirical therapeutics is the use of methods of treatment based upon previous experience.

The dangers that arise in placing reliance on experience is that the number of observations is frequently inadequate, and the conditions under which they are made are so varied that the results obtained in a few uncontrolled observations are unreliable.

Dental therapeutics has made rapid progress in the past decade. In order to develop and promote more rationalized therapeutics, there is need for the wide dissemination of information on drugs which are more specific in their action than those formerly used.

As drugs with a specific pharmacologic or antibacterial action are developed, the employment of previously used remedies of less direct or specialized action should be discontinued. This may be illustrated

## BASIS FOR RATING OF DRUGS

| DISEASE | DRUGS | RATING | | | |
|---|---|---|---|---|---|
| Necrotic Gingivitis | Sodium perborate | | | I | E |
| | Chromic acid | | | I | E |
| | Arsenicals | | | I | E |
| | Dyes | | | I | E |
| | Antibiotics as adjunct | R | | | |
| Local Anesthetics | For routine use: | | | | |
| | High percentage | | | I | |
| | Low percentage | R | | | |
| | For selected cases: | | | | |
| | High percentage | R | | | |
| | Low percentage | R | | | |
| Endodontia | Cresolated formalin | | | | E |
| | Creosote | R | | | |
| | Azochlorimid | R | | | |
| | Monochlorophenol | R | | | |
| | Antibiotics | R | | | ? |
| Periodontal Disease | Oxygen | R | | | E |
| | Caustics | R | | | E |
| | Surgery | R | | | E |
| Pulp Capping | Calcium hydroxide | R | | | E |
| | Zinc oxide eugenol | | O | | E |
| | Copper cement | | | I | E |
| | Gold foil | | O | | E |
| Hemorrhage | Monsel's solution | | O | | E |
| | Tannic acid | | O | | E |
| | Thrombin-containing products | R | | | |
| | Epinephrine | R | | | |
| Post extraction medication | Sulfas | | | | |
| | Dusts | R | | | |
| | Cones | | O | I | |
| | Antibiotics | | | | |
| | Dusts | R | | | |
| | Cones | R | O | | |
| | Anodyne Pastes | R | | | E |

KEY: R = rational / O = obsolete / I = irrational / E = empirical

by the choice of anesthetics for long or short, deep or light anesthesia, or the selection of antibiotics of known bacterial effectiveness for the treatment of specific infections.

Some drugs, which may have had some basis for their use, become irrational and consequently obsolete when more careful biologic and clinical study shows that their irritating or detrimental effects outweigh their possible antibacterial or pharmacologic value. The use of cresolated formalin as in endodontia and the use of sodium perborate in necrotic gingivitis are examples of this type. Another form of irrational therapy is the use of drugs for the treatment of diseases for which they are not indicated. A drug may be a very good drug, and its use rational for the treatment of one disease, but of no value, and consequently irrational, when used in the treatment of another pathologic condition.

It is rational therapy to use drugs which have no value in the cure of the disease but are of value in the treatment of symptoms, pain and discomfort. The use of penicillin in the treatment of necrotic gingivitis illustrates this type.

Table I represents an attempt to evaluate the therapeutic basis of some drugs used in dentistry. Because of the variable conditions under which a drug may be used, there is much overlapping of classifications and justifiable diversity of opinion.

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Associate Professor of Dentistry (Pharmacology and Therapeutics), School of Dentistry, University of Michigan, Ann Arbor, Michigan.

PUBLICATIONS: Dental pharmacology and therapeutics. Endodontics.

HONORS: Omicron Kappa Upsilon and Sigma Xi.
Member of the Council of Dental Therapeutics, A.D.A.
Member of the Advisory Committee to the Bureau of Chemistry of the A.D.A. and Committee on Dentistry of the National Research Council.

## CONTROL OF PAIN: ANALGESICS, SEDATIVES, NARCOTICS

Apprehension and nervousness preceding and during dental appointments is best controlled by the sedative drugs. A member of the barbiturate group is the drug of choice in most instances. The barbiturates are also effective for preventing or reducing the intensity of reactions of a psychological and physiological nature incident to the injection of local anesthetics. On the other hand, the barbiturates are of very little value in the control of pain. Generally speaking, the barbiturates useful for premedication of the dental patient should be rapid in onset, brief in duration, and cause few side reactions. Members of the barbiturate group may be addicting when used excessively. They frequently cause death when taken in over-dosage by accident or with suicidal intent. No patient should be allowed on the street unaccompanied while under the influence of barbiturates because his depressed mental condition may readily lead to accidents.

For the mild to moderately severe pain of dental origin, the analgesics of the antipyretic series are most desirable. Of this group, aminopyrine and acetanilide have potential toxic effects which contraindicate their routine use. Acetanilide is a methemoglobin former, may be addicting, and is sometimes toxic to the heart muscle, while aminopyrine has frequently caused granulocytopenia. On the other hand, acetylsalicylic acid, U.S.P. (aspirin) and acetophenetidin, U.S.P. (phenacitin) are safe and effective when rationally used. Combinations of these two drugs, with a trace of caffeine, are sold under a variety of trade names. While the use of such preparations is not objectionable in itself, they should not be dispensed in such a way that it will encourage self-medication. At one time it was feared the acetophenetidin might cause granulocytopenia,

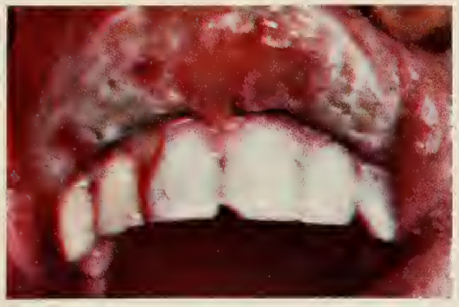

**Fig. 1**
A severe stomatitis which developed in a student as a result of an idiosyncrasy to a member of the barbiturate group

but a recent survey made by the Drug Division of the Food and Drug Administration at the request of the Council on Dental Therapeutics indicates very little possibility of such toxicity from this drug. While acetylsalicylic acid rarely causes toxic effects, a few people cannot tolerate it and urticaria and dangerous acute edema of the respiratory passages and other parts of the body have been reported.

When a more excruciating type of pain occurs in the oral region, due to severe trauma or infection, members of the narcotic group are indicated. Codeine is most adaptable for general use in dentistry but is not as effective in pain relief as morphine. However, the latter drug is not suitable for the ambulatory patient, and it is usually reserved for hospital use. Meperidine hydrochloride (demerol) and methadon are newer synthetic drugs which may have some value in the control of the more severe types of dental pain. To prescribe members of this group the dentist must be registered under the provisions of the Harrison Narcotic Act.

The dentist has a responsibility to himself, his patient, and his profession to control dental pain and apprehension whenever it occurs, with the greatest possible efficiency. Rational use of the sedatives, analgesics, and the narcotics will go far toward the attaining of this end. It will also go far toward eliminating the fear complex which is still inherent in many patients when they approach a dental appointment.

BITURATES

| ONSET TIME | DURATION |
|---|---|
| 15-30 minutes | Short — 3-5 hours |
| 15-30 minutes | Short — 3-5 hours |
| 15-30 minutes | Very short — 1-3 hours |
| 30-60 minutes | Long — 12-24 hours |
| 30-60 minutes | Long — 12-24 hours |
| 20-45 minutes | Medium — 6-24 hours |
| 20-45 minutes | Medium — 6-24 hours |

rotic edema of the lower
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.P. (Aspirin)

**FLOYD D. OSTRANDER,**
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ssor of Dentistry (Pharmacology and The
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: Dental pharmacology and therapeutic

cron Kappa Upsilon and Sigma Xi.
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Advisory Committee to the Bureau of Ch

## CONTROL OF PAIN: ANALGESICS

## DRUGS FOR CONTROL OF DENTAL PAIN

### MILD TO MODERATELY SEVERE PAIN

| DRUG | DOSE | FREQUENCY |
|---|---|---|
| Acetylsalicylic acid | 0.3 Gm. (5 gr.) | 1 hour |
| Acetophenetidin | 0.3 Gm. (5 gr.) | 3 hours |

### SEVERE PAIN

| DRUG | DOSE | FREQUENCY |
|---|---|---|
| Codeine Sulfate | 0.03 Gm. (1/2 gr.) | 3 hours |
| Morphine Sulfate | 8 to 10 mg. (1/8 to 1/6 gr.) | 4 to 6 hours or as needed |
| Meperidene HCl | 50 to 100 mg. | 5-6 hours |
| Methadone HCl | 2.5 to 10 mg. | 3-4 hours |

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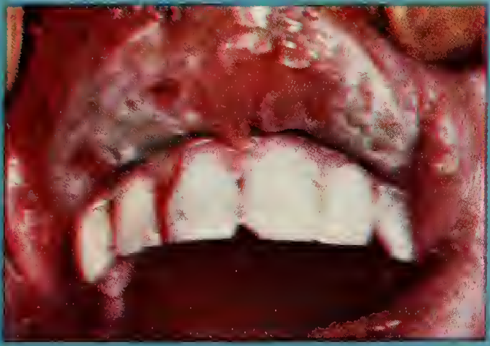

**Fig. 1**
A severe stomatitis which developed in a student as a result of an idiosyncrasy to a member of the barbiturate group

### ONSET TIME AND DURATION OF SOME COMMON BARBITURATES

| DRUG | DOSE | ONSET TIME | DURATION |
|---|---|---|---|
| Pentobarbital Sodium, U.S.P. | 0.1 to 0.2 Gm. (1 1/2 to 3 gr.) | 15-30 minutes | Short — 3-5 hours |
| Seconal Sodium | 0.1 to 0.2 Gm. (1 1/2 to 3 gr.) | 15-30 minutes | Short — 3-5 hours |
| Hexobarbital | 0.13 to 0.39 Gm. (2 to 6 gr.) | 15-30 minutes | Very short — 1-3 hours |
| Phenobarbital Sodium | 0.1 to 0.2 Gm. (1 1/2 to 3 gr.) | 30-60 minutes | Long — 12-24 hours |
| Barbital Sodium | 0.3 Gm. (5 gr.) | 30-60 minutes | Long — 12-24 hours |
| Amobarbital Sodium (Amytal) | 0.1 to 0.2 Gm. (1 1/2 to 3 gr.) | 20-45 minutes | Medium — 6-24 hours |
| Butethal (Neonal) | 0.1 to 0.4 Gm. (1 1/2 to 4 gr.) | 20-45 minutes | Medium — 6-24 hours |

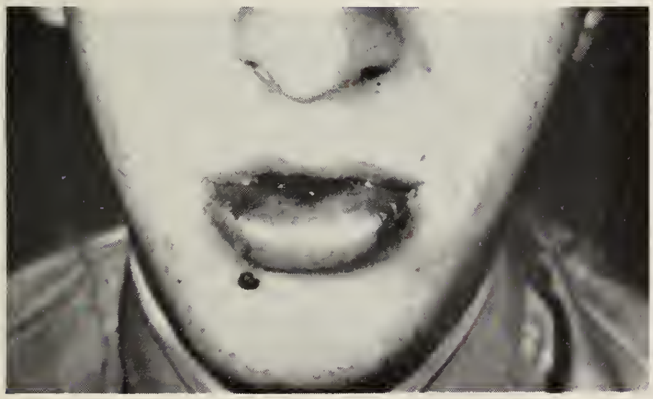

**Fig. 2**
Angioneurotic edema of the lower lip. Reactions of this type as well as other serious toxic manifestations have been reported occasionally following the use of acetylsalicylic acid U.S.P. (Aspirin)

**C. PHILLIP MILLER, M.D.**

Professor of Medicine, University of Chicago.
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PUBLICATIONS: Numerous papers in the field of infectious diseases, bacteriology and the antibiotics.

HONORS: Medical Fellowship Board of National Research Council, Association of American Physicians, American Society for Clinical Investigation, (Emeritus), History of Science Society, Society for General Microbiology (Great Britain).

# CONTROL OF INFECTION: ANTIBIOTICS AND SULFONAMIDES

The control of infection in the oral cavity and its adjacent structures has been greatly facilitated by the introduction of sulfonamide drugs and the antibiotics. The proper use of these chemotherapeutic agents requires an understanding of their mode of action and an appreciation of their limitations and of their possible danger to the patient.

The therapeutic effectiveness of all of these drugs depends upon their ability to kill or to inhibit the multiplication of certain species of bacteria. Fortunately, most of the bacteria which produce infections for which the dentist is responsible are susceptible to the action of *penicillin, aureomycin, chloramphenicol (chloromycetin)* and the *sulfonamides*.

To be effective, any of these drugs must be brought into direct contact with the bacteria, usually by way of the blood stream because most of the infections which require the dentist's attention are situated beneath the surface. The drug must, therefore, be administered in such a way to insure absorption by the blood. All of those mentioned above can be given by mouth, but penicillin is economically administered by intramuscular injection. Local application is effective in only the most superficial infections because the drug is unable to penetrate into inflamed tissues sufficiently to build up an effective concentration. The capillary circulation carries it away too rapidly.

One very important use of the antibacterial drugs in dentistry is in the prevention of bacterial endocarditis.

Proper use of these antibacterial agents requires: (a) correct bacteriological diagnosis of the infection, (b) the selection of the most effective drug for its control, and (c) consideration of the possible hazards involved in such medication. All of these drugs may cause sensitization of the

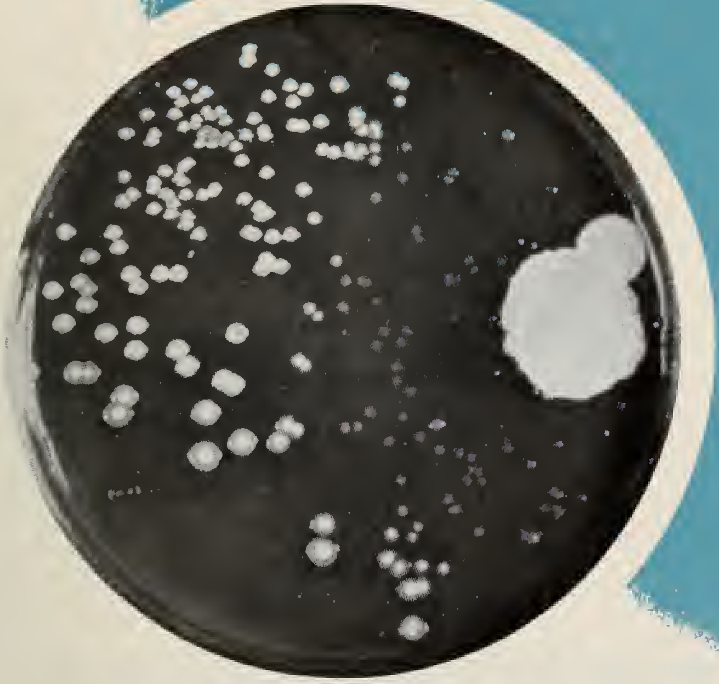

**Fig. 1**
(From Fleming) A photograph of the original plate on which Fleming found a colony of *Penicillium* dissolving the surrounding colonies of Staphylococci. This was the first observation of the action of what was subsequently called penicillin. (Courtesy W. Florey, Antibiotics Vol. I, 1949)

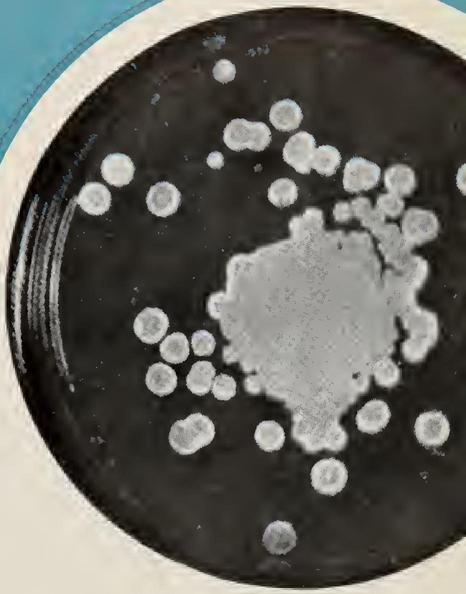

**Fig. 2**
Streptomyces griseus (Courtesy S. Waksman, Rutgers University)

patient with resultant development of urticaria or other manifestations of hypersensitivity. The sulfonamides may cause a variety of toxic effects such as hematuria or even anuria. This can be avoided if the urine is kept alkaline.

The long continued administration of these drugs should be discouraged because:

1. the possibility of producing a state of hypersensitivity in the patient;
2. it may result in the development of resistant strains of bacteria; and
3. it may adversely disturb the normal bacterial flora of the mouth.

Although modern chemotherapy provides a highly valuable weapon against infection, it cannot supplant the practice of sound dentistry.

**J. GARROT ALLEN,**
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HONORS: Society for Experimental Biology and Medicine, American Physiological Society, Society of University Surgeons, Gold Medal American Medical Association, 1948.

## CONTROL OF BLEEDING: HEMOSTATICS

The mechanism of hemostasis is complicated and poorly understood. Abnormal bleeding, either spontaneous or traumatic, may arise from any disturbance involving the coagulation of blood, the platelet concentration of plasma, or the permeability of the walls of small vessels. At times the cause of hemorrhage is singular, but more often multiple factors are involved. The diagnosis of abnormal bleeding depends upon a complete analysis of those factors already known to affect the hemostatic mechanism. It must be clearly recognized that there are probably a number of factors which are not yet known and contribute to bleeding.

**Prothrombin reduction.** Perhaps the most easily recognized deficiency of coagulation is a reduction in prothrombin activity. This is readily corrected by the parenteral administration of adequate amounts of suitable preparations of vitamin K when due to inadequate absorption. It may also result from the inability of the liver to produce prothrombin even though adequate vitamin K is made available to it. Prothrombin may be passively transferred by blood or fresh plasma transfusions. There is indication that there may soon be available a human preparation of purified prothrombin which may be injected when bleeding is imminent. The effect of transfused prothrombin, however, is relatively brief, generally less than six to eight hours. Repeated transfusions at the appropriate intervals are necessary to prevent further bleeding.

**Fibrinogen deficiency.** The congenital absence or severe reduction of fibrinogen has been reported by several investigators. Here again the incidence of this disorder is very infrequent. Several of the patients reported have lived to ten to fifteen years of age. If these patients represent a true congenital absence of fibrinogen, it is indeed surprising

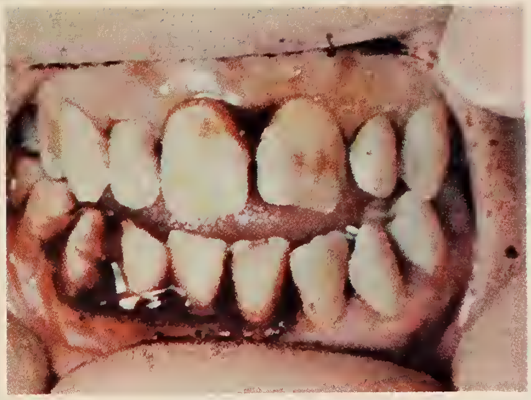

**Fig. 1** Patient with acute myelogenous leukemia and extensive gingival bleeding.

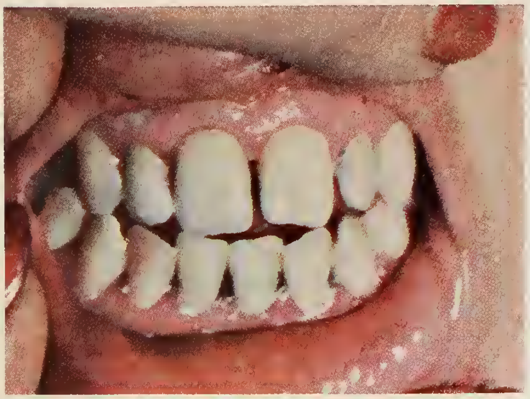

**Fig. 2** Condition of mouth of patient in Fig. 1 twelve hours following intravenous administration of 300 mgm. toluidine blue.

that the infant can survive the process of birth and its attendant trauma. The passive transfer of fibrinogen by transfusion is effective in the control of bleeding in such patients. During the bleeding episode, frequent transfusions are necessary.

**Hemophilia.** The nature of the fundamental disturbance in true congenital hemophilia is not yet known. These patients respond to some extent to whole blood and plasma transfusions or to antihemophilic globulin. These agents produce an incomplete and short-lasting effect. Surgery should not be advised in the hemophilic patient, except under a most urgent condition. Even then, the risk is great and many patients die in the early postoperative period from uncontrollable internal bleeding.

**Idiopathic thrombocytopenic purpura.** This disorder was described more than 200 years ago as a clinical syndrome. It was not recognized as being associated with platelet deficiency until 1883. Splenectomy was first suggested for its treatment nearly thirty-five years ago. The results have been surprisingly good in some cases and disappointing in some others. As hemocytologists have become better acquainted with the morphology of the bone marrow, it has been possible to predict with a higher degree of accuracy the patients that are suitable candidates for splenectomy. When the diagnosis has been made and the patient is suffering from severe bleeding, the removal of the spleen becomes an emergency procedure. Intracranial hemorrhage is a frequent fatal complication in the untreated patient. Thus far, no other effective therapy has been devised. Transfusions are of value only in correcting the anemia and are of little or no value as a means for increasing the platelet concentration.

Some of the patients with thrombocytopenia, whether idiopathic or otherwise, suffer from another defect in the hemostatic mechanism. This is detectable by a carefully performed clotting time and by a protamine titration. The defect resembles that produced by heparin in certain respects, but does not appear to be the result of a substance of a clotting inhibitor identical with commercial heparin. When these coagulation abnormalities can be demonstrated, protamine sulfate or toluidine blue will decrease the tendency to bleed to the extent that this clotting disorder contributes to the over-all hemorrhagic picture. The nature of this hemostatic disturbance is not known. While the defect resembles that produced by heparin, it may also be produced by other mechanisms and agents not related to heparin.

Unfortunately, there is no simple reliable method for determination of the whole blood clotting time. When careful technique is employed, the normal values in clean glassware approximates twenty-five to forty minutes for the human subject. Techniques currently employed in most laboratories run from 3 to 10 minutes and represent maximum acceleration of coagulation. Methods of this type therefore are of little value.

**Capillary permeability.** An increased leakage of blood from the smaller vessels may occur as the result of an increased permeability of the capillaries or from a decreased resistance of the smaller vessels to intravascular pressure. A number of agents are said to influence capillary permeability, but the results obtained are not in general agreement.

The hemostatic mechanism is the first response of the organism in wound healing. It is concerned not only in patients with abnormal bleeding but in many other physiological processes that confront not only the hemocytologist but the doctor in general practice as well. Until more is known of this difficult subject, therapy for abnormal bleeding will remain inadequate and incomplete.

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# HAZARDS OF IRRADIATION: THERAPY ON ORAL TISSUES

Radiant energy has a variety of applications. It affects human tissue by the production of heat and by the chemical action on living cells.

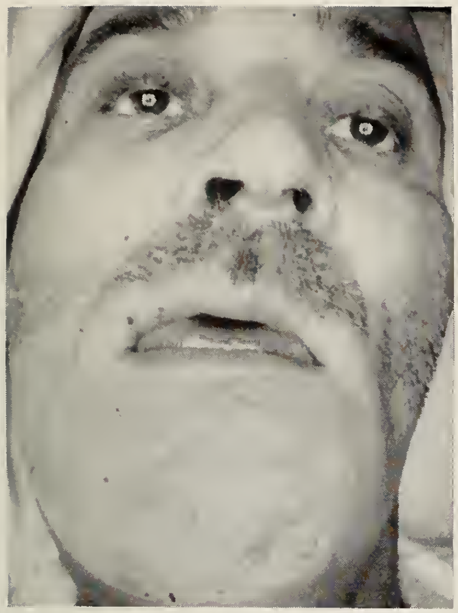
Heat is produced by the infra-red rays and by diathermy. The infra-red radiation produces deep heat, and that produced by diathermy penetrates still more.

Chemical action on living cells is produced by ultraviolet radiation and roentgen rays. Ultra-violet radiation has a tonic and stimulating action and produces an erythema. It is supposed to have a bacteriostatic effect. Since the advent of antibiotic therapy, the latter is of no great importance for practical therapeutic use.

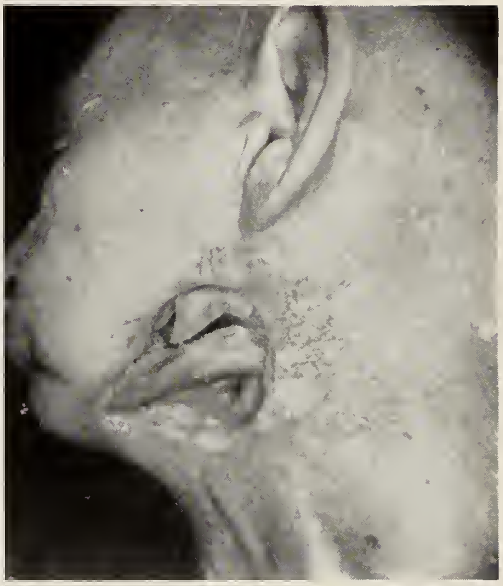
The roentgen rays and the gamma rays of radium are the most important of these agents, not only because their effect is more easily recognized, but mainly because they may be employed for the cure or arrest of malignant diseases. Irradiation with these agents produces a growth restraining, autolytic, necrotizing effect. The result, however, is subject to great variations. Not only the tumor tissue but also normal structures are affected. Some tissue cells are more readily affected than others, undifferentiated cells succumb more easily than highly differentiated ones.

Dental patients who have received irradiation therapy for oral lesions must receive special consideration by the dentist. Dental infection or peridontal disease may lead to serious irradiation osteomyelitis. Ewing has shown that bone irradiated during treatment of a malignant lesion undergoes necrosis.

If x-ray treatment is given, the normal mouth is not safe if teeth are present, since infection of the marginal gingiva occurring during the



ceived X-ray irradi-
f floor of the mouth,
necrosis and sub-
lote absence of hair
egion.



resulting epithelitis may be sufficient to start this distressing bone disease. The suffering from these complications is so great and the deformities resulting are so disfiguring that in most cancer clinics the extraction of all teeth or at least those in the path of the rays is advised before irradiation is started. Some of the teeth may be saved by the use of protective shields.

If teeth are allowed to remain, irradiation caries may set in, which are cervical erosions that progress to the point of amputation of the crown of the tooth. The care of such teeth to prevent the necessity of extraction, the prevention of ulcerations caused by prosthetic appliances, and the treatment of resulting infections is an important subject, and in many cases xerostomia, which causes considerable suffering, must be dealt with.

An additional disadvantage to the use of irradiation is that this treatment at once eliminates future surgical procedures for recurrence of cancer or to obtain a better cosmetic result. I prefer surgery, which should be radical when first used, or electro-coagulation, which causes only local necrosis if bone is involved and reserve irradiation for special situations in which surgery is contraindicated.

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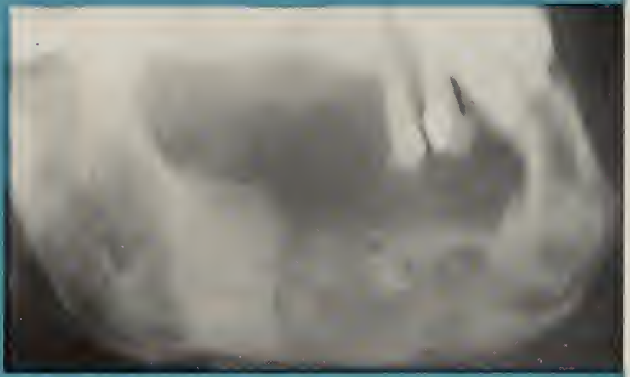
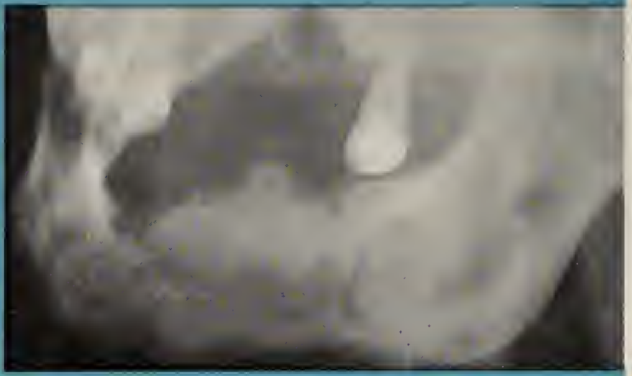
# HAZARDS OF IRRADIATION: TH



**Figs. 3-4**
Roentgenograms showing irradiation necrosis and osteomyelitis in patient shown in Fig. 1.



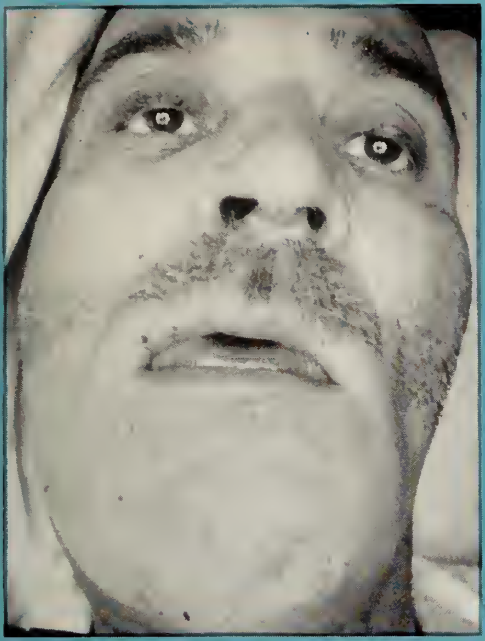


**Fig. 1**
A patient who has received X-ray irradiation for carcinoma of floor of the mouth, resulting in radiation necrosis and submandibular abscess. Note absence of hair growth in irradiated region.

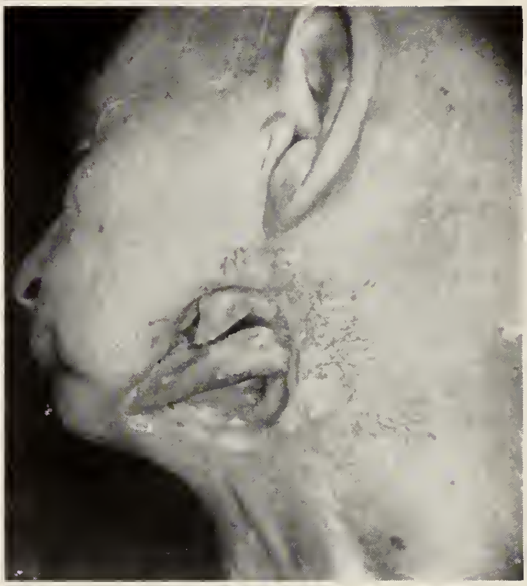


**Fig. 2**
Patient had received heavy radiation therapy for carcinoma. Ensuing hemorrhage required ligation of external maxillary artery. The operative procedure resulted in slough of the skin, exposing the necrotic bone. Note fracture in ramus.

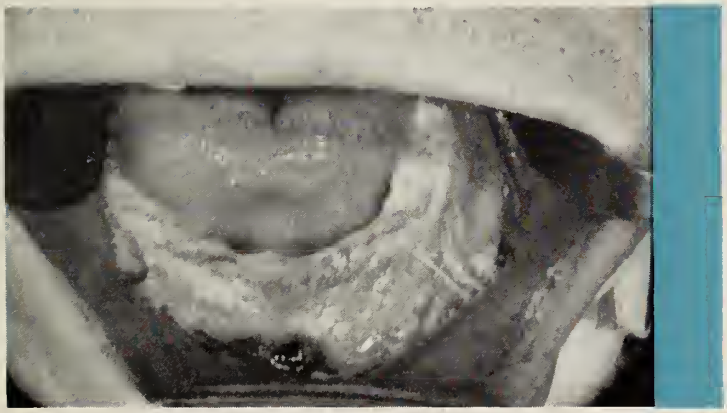


**Fig. 5**
The entire anterior part of the mandible exposed and sequestrated following irradiation.

# NOTES

## QUESTIONS

## NOTES

### RATIONALE OF DRUG THERAPY

1. Is empirical therapy ever justified?
2. Is it possible to use drugs which have a rational basis in an empirical manner?
3. What are the determining factors which make established methods of therapy obsolete?
4. When a drug has been established as having known therapeutic value for the treatment of a systemic disease, is it irrational to use it in the treatment of a similar oral disease?
5. What is the rationale of symptomatic treatment?

### CONTROL OF PAIN

6. What precautions should always be observed when a patient is premedicated with a barbiturate?
7. Should barbiturates be used routinely before administration of local anesthetics?
8. In case a patient cannot take barbiturates, is there any drug or drugs which may be substituted?
9. Is there a rational basis for the combination of barbiturates and antipyretics, such as aspirin, for pain relief?
10. Are there dangers in connection with continual self-medication with the analgesic drugs?
11. Is there any significant difference in the margin of safety in the various members of the barbiturate group?

### CONTROL OF INFECTION

12. Which of the antibacterial drugs are most useful in Dentistry?
13. Which of these are the least toxic?
14. What harmful effects can even the least toxic drugs produce in a patient?
15. Why is topical application seldom effective?

NOTES

## QUESTIONS

16. Why is it important to know what microorganism is causing an infection?

17. What can you do to prevent your patient with valvular heart disease from developing bacterial endocarditis?

18. Why is the long continued use of antibiotics inadvisable?

### CONTROL OF BLEEDING

19. What procedures can be used to determine so far as possible whether the patient has an abnormal bleeding tendency?

20. To what extent does infection contribute to bleeding?

21. Does vitamin C have any effect on the clotting mechanism?

22. What effect has saliva on coagulation?

23. Can anything be done for oral bleeding in the patient with blood dyscrasias?

24. What conditions of bleeding contraindicate oral surgery?

25. Are there agents which may be applied locally to patients with oral bleeding?

### HAZARDS OF IRRADIATION

26. Should the dentist give or prescribe the doses of irradiation for malignant oral disease?

27. How does xerostomia resulting from irradiation affect the teeth?

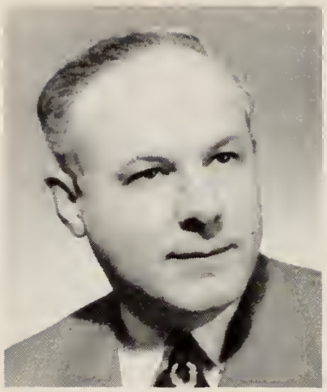
28. Is irradiation beneficial in cases of cellulitis of the face and neck?

29. In what types of cases is x-ray irradiation to be preferred to surgery?

30. What is the difference between low voltage irradiation and radiation with a million volt machine?

31. What is the difference between x-ray and radium therapy?

# NOTES



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# PERIODONTIA

**Monday, December 11, 1950**

## OUTLINE

1. Progress in Periodontia..........Saul Levy
Moderator
2. Classification and Diagnosis of Periodontal Disease..........Balint Joseph Orban
3. The Problem of Reattachment..........Henry M. Goldman
4. Subgingival Curettage—Home Care..........Dickson G. Bell
5. Surgical Treatment of Periodontal Disease..........Olin Kirkland
6. Occlusion and Its Relation to Periodontal Disease..........Samuel Charles Miller

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Miller, Samuel Charles, 1943, *Textbook of Periodontia,* Blakiston's Son and Company, Inc., Philadelphia, Pa.

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## SUMMARY

**PROGRESS IN PERIODONTIA.** In the adult, periodontal disease is responsible for the loss of more teeth than is dental decay. Gingival and periodontal disease has, therefore, been considered a major dental disease second to dental caries. However, with the promising advances in caries control, the periodontal disease may well become the major challenge in dentistry unless equal and even more attention is paid to its control. The teeth saved from destruction by dental decay are still exposed to the influences that lead to periodontal injury. Thus, the saving of the teeth from decay only to be lost by periodontal disease is but an empty victory.

Fortunately, evidence is accumulating to show that periodontics which started as an empirical technique is developing into a science. This evolution of periodontology is mainly due to the extensive biologic, histopathologic and chemical research which has been conducted in the last thirty years.

Gingival biopsies have paved the way for a scientific correlation between the clinical appearance of the gingiva and the microscopic structure. This method has opened up new vistas on the possible etiologic factors of gingival disease, on the normal histophysiologic characteristics of the gingiva and on the characteristic changes with age.

Experimental studies are beginning to reveal the mechanism by which calculus, food debris and other local factors and irritations produce gingival and periodontal lesions. The problem of the role of the physical character of the diet on the health of the gingiva is also being investigated.

Another research attack which will place periodontia on a more scientific basis is the development of methods of assessment of gingival disease so that it may be measured in amount and severity in different population groups.

These and other investigations, too numerous to mention, are a mark of the increasing maturity of dentistry as a science. They are paving the way for scientific advances toward the better understanding of the nature of periodontal disease, toward a more rational basis of diagnosis and treatment and toward its eventual prevention.

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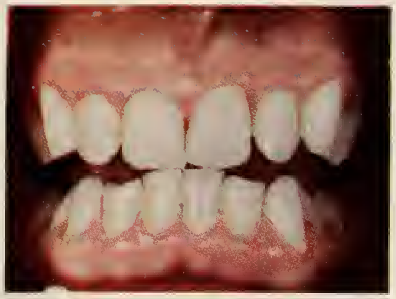
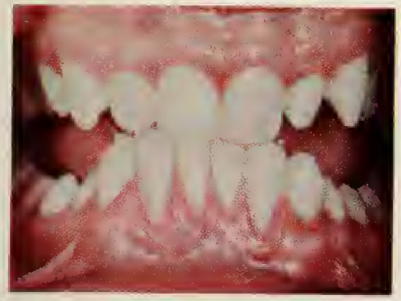
# CLASSIFICATION AND DIAGNOSIS OF PERIODONTAL DISEASE

The scientific achievements in the field of periodontia permit us today to undertake a classification of periodontal diseases based upon pathologic tissue changes, clinical features, and roentgenologic interpretation of the etiologic factors at work. (Refer to Supplementary Chart on Classification of Periodontal Diseases).

The classification as presented in the Supplementary Chart seems at first glance, to be complicated. However, for those who keep up with developments in dental science, it soon will become clear that this classification is quite simple and the clinical application of the data in this chart will enable the practitioner to fully realize that the periodontal disturbances are manifold. There is not one disease, "Pyorrhea" or "Periodontoclasia" of our predecessors, but there are several groups of diseases, different from each other in their tissue changes, clinical and roentgenologic findings and etiology. Naturally, there must be a different approach to their treatment. Many failures in the treatments in the past can be attributed to the lack of understanding of the basic principles involved. However, we are not at the end of the road. We cannot even take a rest. This classification of diseases is only a challenge, in that clarification of detail is now needed. A classification is not a permanent structure; it is only a "filing cabinet". With advancements in clinical and basic scientific investigations, it can be expanded or reduced as the needs arise.

Some men might have the opinion that a classification is not necessary. At least, such opinions have been voiced at different times. In the previous paragraph, one of the reasons was discussed. There is another reason in favor of a classification—for scientific progress. Frequently a periodontal investigator is joined by a research man who has no dental





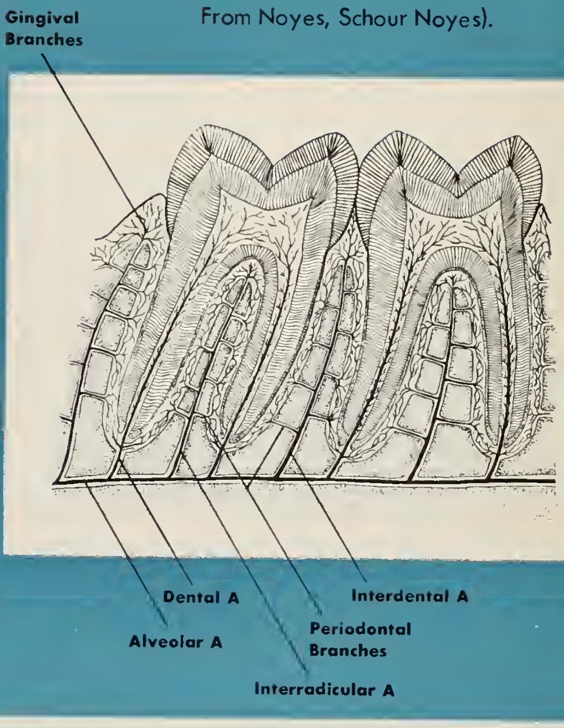
or periodontal training, and depends entirely upon the clinician's observations. If the clinical diagnosis is inaccurate and is termed just "periodontoclasia", the laboratory investigation becomes valueless. Unfortunately, such was and is the case in numerous instances.

Some men might think that a classification should be based primarily on etiologic or topographic factors, upon the acuteness or chronicity of the disease or, only upon the clinical features. All such classifications might have advantages and disadvantages.

The classification presented in the chart takes most of these factors into consideration with full understanding of the difficulties.

**Fig. 5**

Diagram of the distribution of the branches of the Alveolar Artery. (Courtesy H. Sicher, From Noyes, Schour Noyes).



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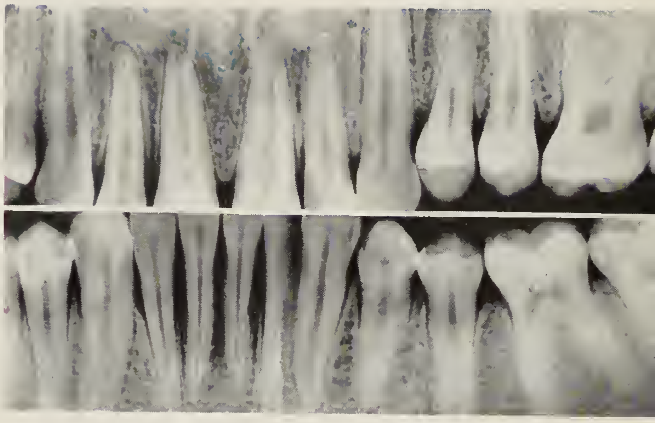

**Fig. 3**

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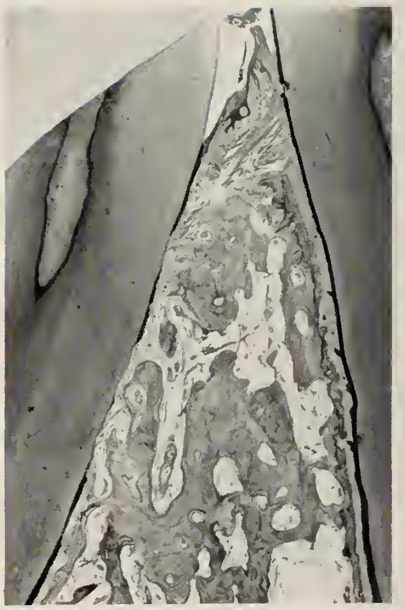
## CLASSIFICATION AND DIAGNO AL DISEASE

**Fig. 4b**
Microscopic specimen of case shown in Fig. 4a. Serious traumatic damage to periodontal membrane, bone and cementum on mesial side of 2nd bicuspid (corresponding to widening of space in roentgenogram). No pathologic changes in the gingiva.



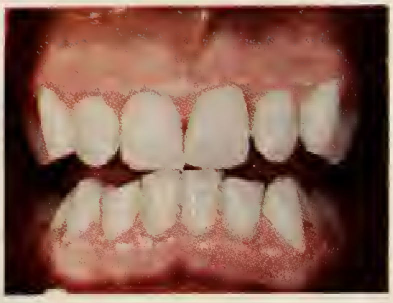


**Fig. 1**

Patient, Age 25
Extensive overbite
In protrusion, only two lower incisors in occlusion. No damage to supporting tissues.

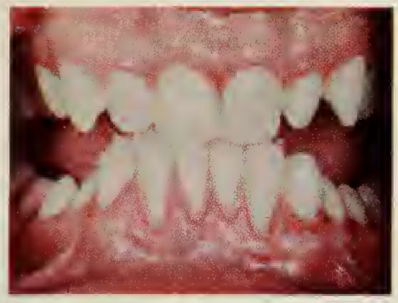
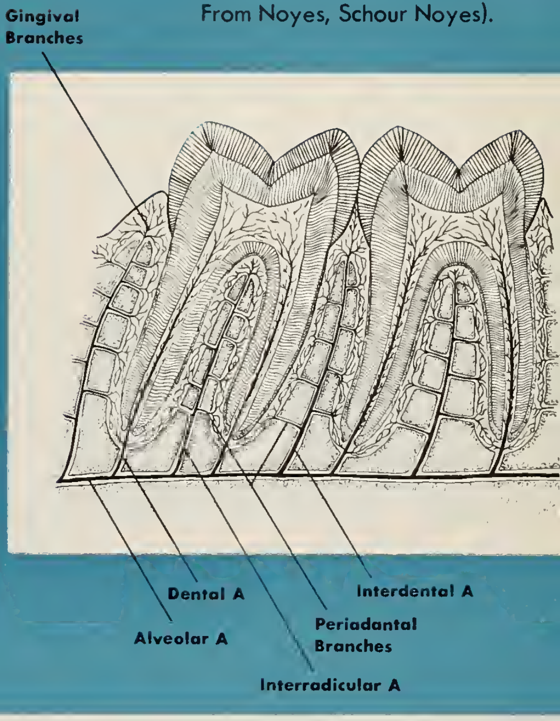


**Fig. 2**

Patient, Boy, Age 16
Extensive overbite—Damage to labial gingiva due to shearing off of tissues by upper incisors.

**Fig. 5**

Diagram of the distribution of the branches of the Alveolar Artery. (Courtesy H. Sicher, From Noyes, Schour Noyes).



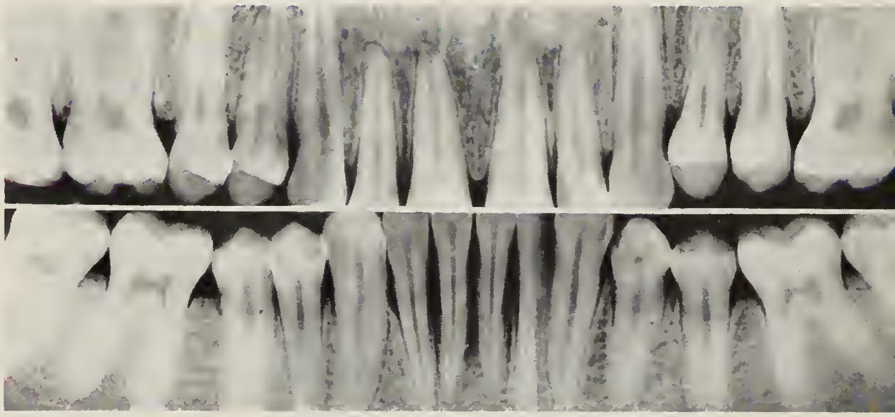


**Fig. 3**

Roentgenograms of case shown in Figure 2
No loss of alveolar bone in any other than labial area of lower anteriors which x-ray does not show.

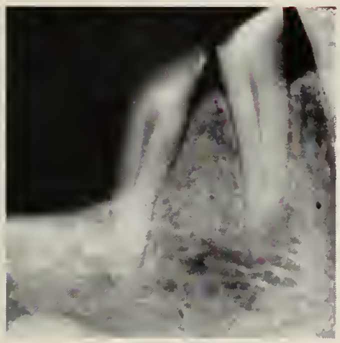


**Fig. 4a**

Roentgenogram of two lower bicuspids
Occlusal wear—Widening of periodontal space on mesial side of 2nd bicuspid.

**b2**

PERIODONTIA

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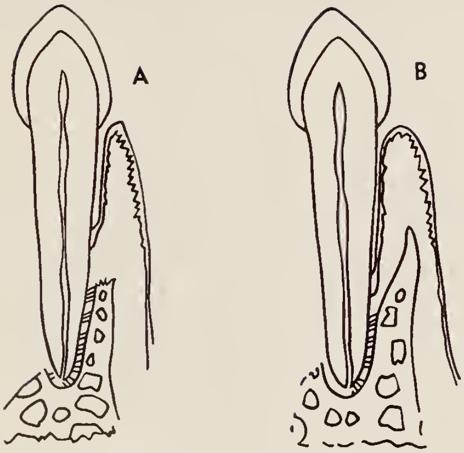
## THE PROBLEM OF REATTACHMENT

The problem of reattachment of detached gingival tissues to the root surface has been discussed for years and as yet no clear-cut evidence has been presented to settle this question. It is essential, first of all, to define the term "reattachment". The problem of reattachment must be further considered according to the type of pocket, which may be classified as gingival (A) or intrabony (B). Thus the process of reattachment may be described as follows:

1. A reestablishment of an organic connection between tooth and gingival corium. This calls for production of cementum and organization of gingival and transeptal fibers of the periodontal membrane.

2. A reestablishment of an organic connection between the tooth and bone. This calls for production of cementum, reestablishment of a periodontal membrane, and apposition of bone.



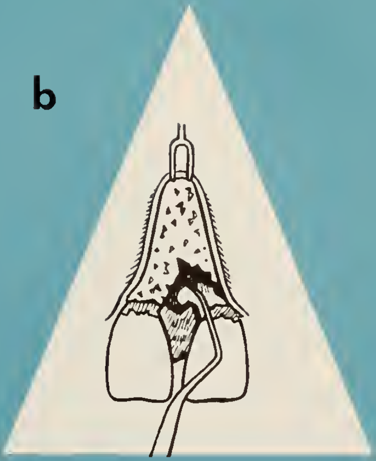
**Gingival Pocket.** (A) In the gingival pocket the base of the pocket is coronal to the alveolar crest. True reattachment and not merely close apposition of the pocket epithelium of the tooth surface must consist of the removal of the entire pocket epithelium as well as the epithelial attachment. Allow a blood clot to form and organize with subsequent reestablishment of the normal functional anatomy of the area. From a physiologic point of view this is certainly possible. As yet no definite proof of reattachment in this type of pocket has been offered.

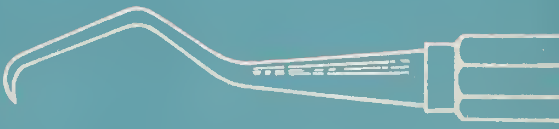
**Intrabony Pocket.** (B) In the intrabony pocket, the base of the pocket is apical to the alveolar crest. Clinical reports indicate that intrabony pockets can be eliminated by means of a true reattachment of the principal fibers with new formation of cementum and bone. The formation of these tissues depends upon the organization of a blood clot between the tooth surface and the bone, just as occurs following root canal therapy and apicoectomy for a periapical lesion. It has been shown that the apical surface of the tooth is covered with cementum after these procedures, that there is a regrowth of an organized periodontal membrane and that bone is deposited on the alveolar side. Many reports attest to the fact that bone apposition (with regeneration of a lamina dura) occurs after therapy of an intrabony pocket.

**Clinical Rationale:** In the treatment of the gingival pocket, the choice of procedure depends on the periodontal condition. Shallow gingival pockets are best treated by sub-gingival curettage. A healthy gingival sulcus will result after healing of the pocket epithelium and resolution of inflammation. Pockets of moderate depth associated with swollen, edematous, spongy gingivae can also be successfully treated by subgingival curettage. However, subgingival curettage will not be effective in the treatment of deep pockets with long-standing fibrotic inflammatory gingival hyperplasia. Such pockets are best treated by gingivectomy.

The operator has two choices in the treatment of a pocket in which the base is apical to the bone crest. First, he can attempt to produce reattachment; and, second, he can remove the bone crest to a level of that of the base of the pocket. Otherwise, he must remove the tooth in order to eliminate the pocket.

b





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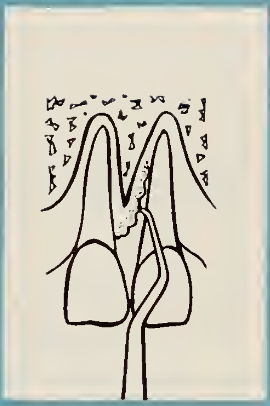

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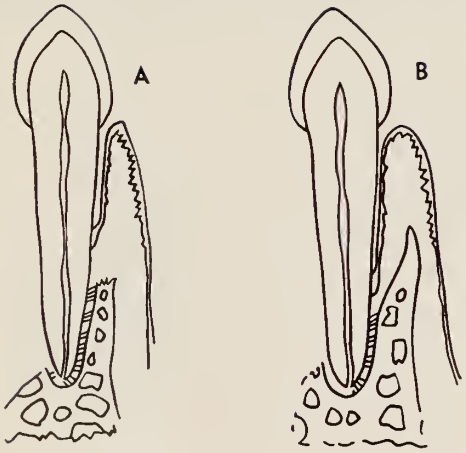
HENRY M. GOLDMAN
D.M.D

# THE PROBLEM OF REATTAC



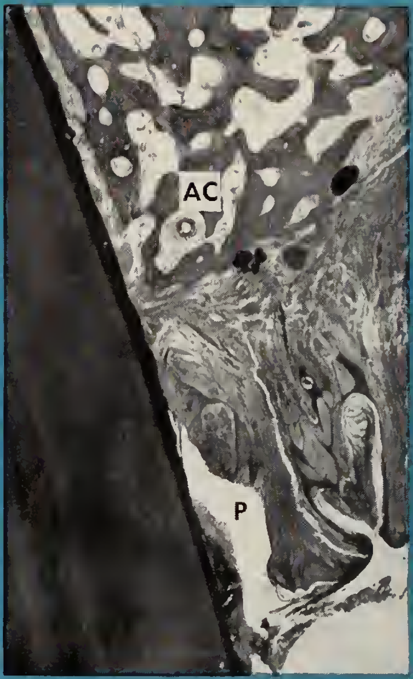


**Fig. 1**
Photomicrograph of a gingival pocket. Note that the base lies crown-wise to the alveolar crest. (P, pocket; AC, alveolar crest)

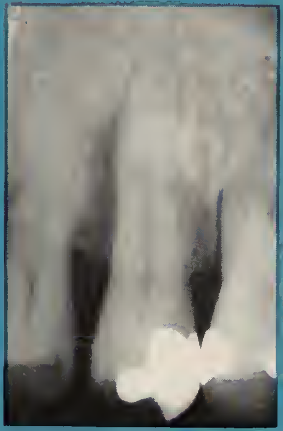


**Fig. 2**
Roentgenogram of an intrabony pocket. The vertical resorptive lesion can be seen. An explorer can be passed into the pocket far beyond the alveolar crest.

his is certainly possible. As yet no definite
type of pocket has been offered.

he intrabony pocket, the base of the pocket is
t. Clinical reports indicate that intrabony
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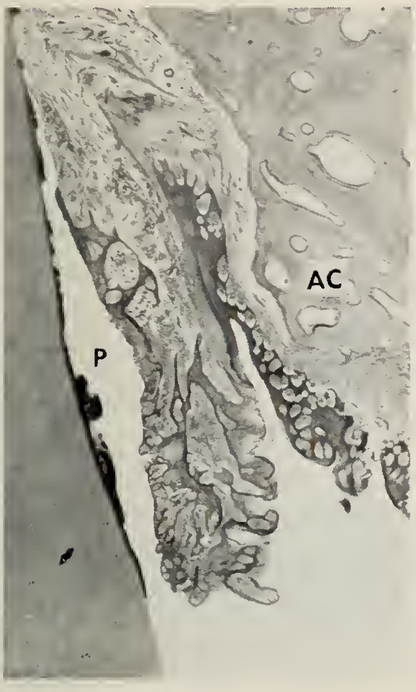
mation of cementum and bone. The formation
on the organization of a blood clot between

**Fig. 3**

Photomicrograph of an intrabony pocket. The base of the pocket is situated root-wise to the alveolar crest. (P, pocket; AC, alveolar crest)



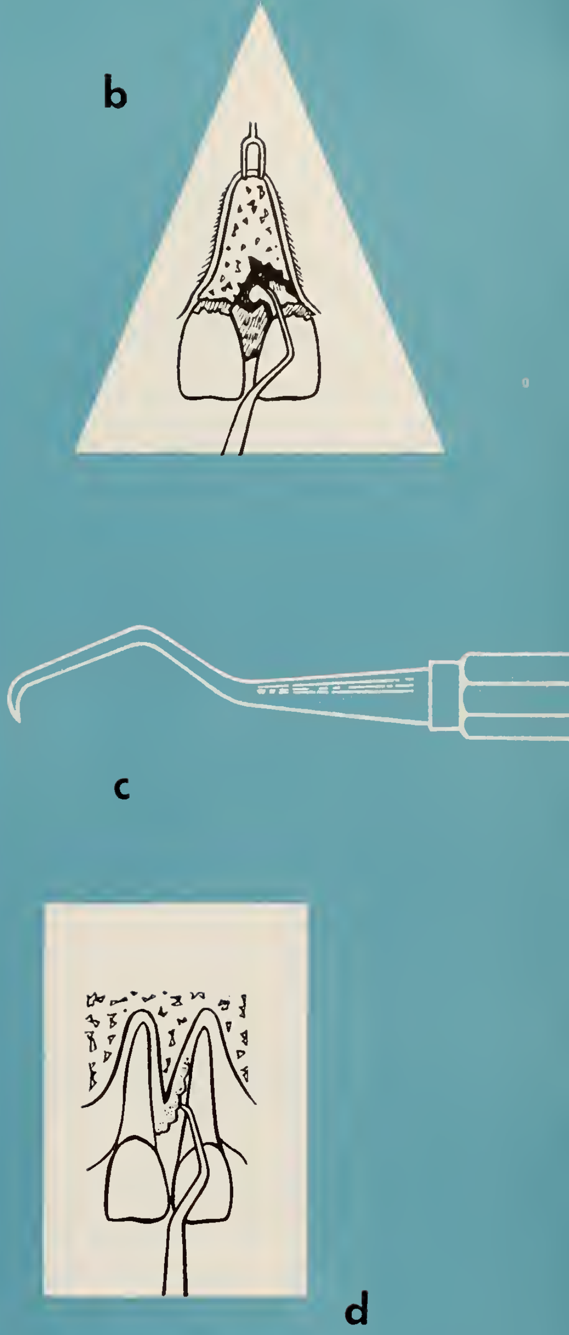
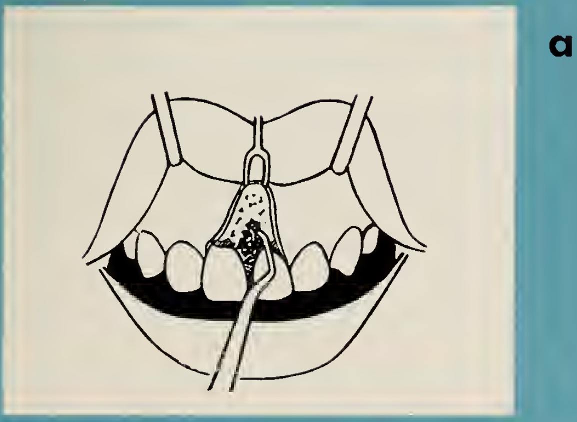


**Fig. 4**

Diagram showing therapy of an intrabony pocket. Not only is the pocket epithelium together with the epithelial attachment removed, but also curettage of underlying tissue is done. The free gingival tissue is excised.



**DICKSON G. BELL,
D.D.S.**

Practice limited to Periodontia, San Francisco; Department of Periodontia, University of California College of Dentistry, San Francisco, California.

PUBLICATIONS: Contributor to the literature in the field of Periodontia.

HONORS: Omicron Kappa Upsilon, President, American Academy of Periodontology.

## SUBGINGIVAL CURETTAGE

There has been much discussion concerning the objective of successful treatment of the periodontal lesion and the procedure by which it may be obtained. The majority of workers in this field agree there should be a reduction of the depth of the pocket. Either by means of contraction of the edematous gingiva from repair within the lesion or by surgical removal of the soft tissue.

Some periodontists feel that following therapy the depth of the gingival sulcus should be 0 to 1 mm. Others are of the opinion that a sulcus of greater depth can be maintained free of inflammation by periodic prophylaxis and proper home care. The purpose of treatment is to stop the progress of the disease and to maintain the teeth and their supporting tissues in functional equilibrium for as long a period as possible. This should be brought about by a therapeutic procedure which will resolve the inflammatory process and reduce the pocket to a minimal sulcus, without unnecessary loss of soft tissue or alveolar bone.

The type of local treatment selected is dependent on the character of the lesions about the individual teeth and upon the over-all diagnosis. In every case it is advisable that conservative measures should be given first consideration. The results obtained by subgingival curettage are such that in many cases it is the only procedure necessary. Subgingival curettage in the other cases removes the local irritating factors, thereby reducing the inflammatory reaction and preparing the tissues for further surgery. All methods of therapy have advantages and disadvantages and only through a knowledge of tissue reaction and healing potential can a sense of judgment be developed. A correct evaluation of all local and systemic factors is essential to good periodontal procedure.

Subgingival instrumentation should be done systematically. The teeth are treated in a continuous order. In this way there is no interference with tissue healing in the previously treated areas. The curettage around one tooth should be thoroughly finished before proceeding to the next. Treat only as many pockets as you can complete at one appointment. Effective subgingival curettage depends on the degree of thoroughness with which the instrumentation is performed. The blood plays a very important role in curettage since the blood clot is essential for repair of the tissues.

## HOME CARE

Complete instruction in proper home care is also a very important therapeutic procedure. The ultimate success or failure of the treatment of the periodontal lesion depends to a large degree on the cooperation of the patient. Clinical observation has shown that the majority of patients do not know how to properly care for their mouths because they have received no instruction from their dentists. The many different designs and bristle textures of toothbrushes today lead the patient to assume that it does not make much difference which one he uses.

The most satisfactory type of brush is one that has a flat or level trimmed bristle surface—the so-called straight toothbrush. It should have a large number of bristle knots or tufts set close together and four rows in width. This gives a degree of density or body to the brush. The many bristles will cover more area of the tooth surface and thus give a better cleansing result. The bristles should not be too stiff and the brush of medium size so as to reach all areas of the mouth.

Good mouth hygiene means not only the correct use of the toothbrush but also thorough cleansing of the interproximal areas. Therefore, patients should also be given specific instructions in this procedure. The agents used for this purpose are dental floss, dental tape, soft wood points and soft rubber cones. Each has its particular indication, depending on the height of the septal gingiva or size of the interproximal space and should be suitably prescribed by the dentist.

The principles of correct home care are discussed with the patient during periodontal treatment. The actual demonstration in the mouth and the patient's application of the principles are deferred until the inflammation has been reduced and tissue repair has taken place. The patient should not be dismissed until he has demonstrated his ability to properly care for his teeth and investing tissues.

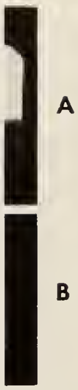


**Fig. 1a**

Periodontal case treated over 22 years ago by subgingival curettage. Comparison of 1950 pictures (B) with those of 1928 (A) shows no further bone resorption and restoration of the cortical plate may be noted in many areas.

**Fig. 1b**

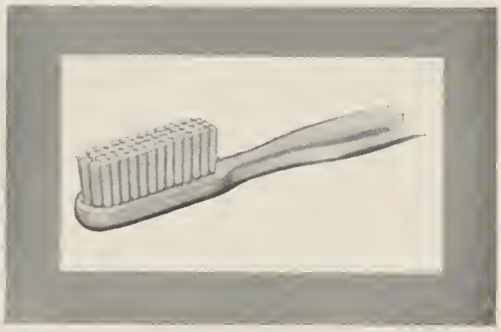


**Fig. 4a**

A four row level trimmed toothbrush.

**b4**

PERIODONTIA

**DICKSON G. BELL,**
**D.D.S.**

# SUBGINGIVAL CURETTAGE

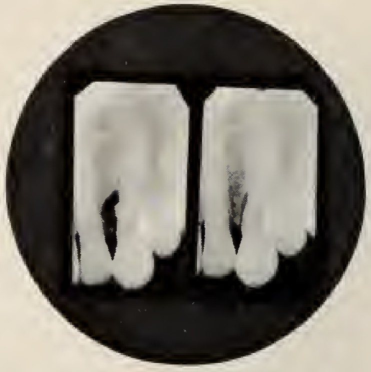
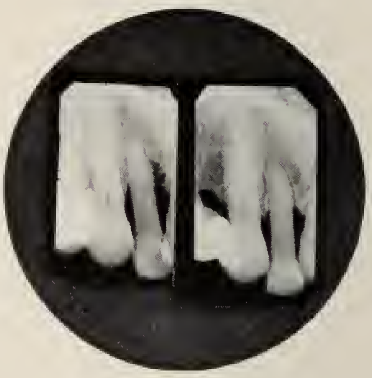


**Fig. 2a**
Illustration of bone repair (right) following subgingival curettage.
**Fig. 2b**



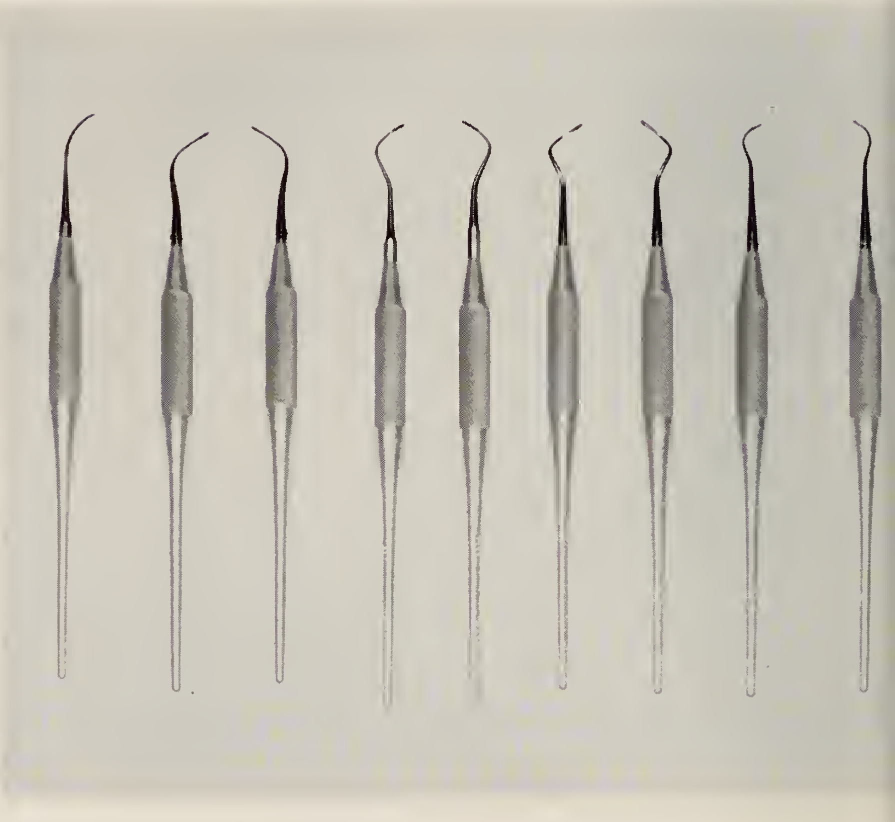
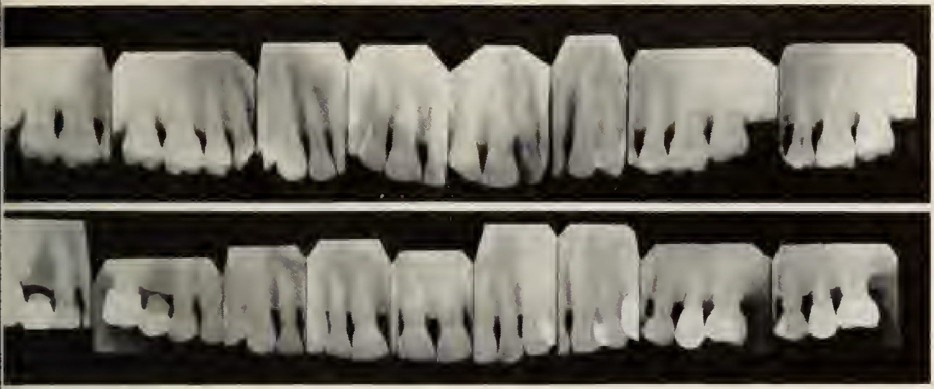


**Fig. 3**
Set of instruments used for subgingival curettage.



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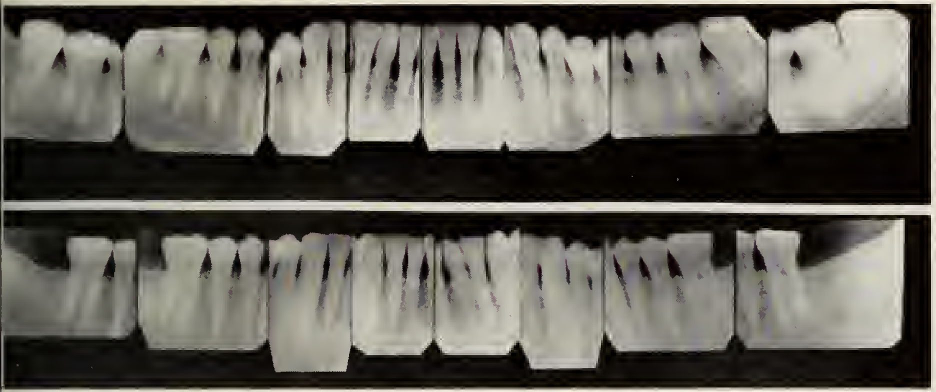


**Fig. 1b**

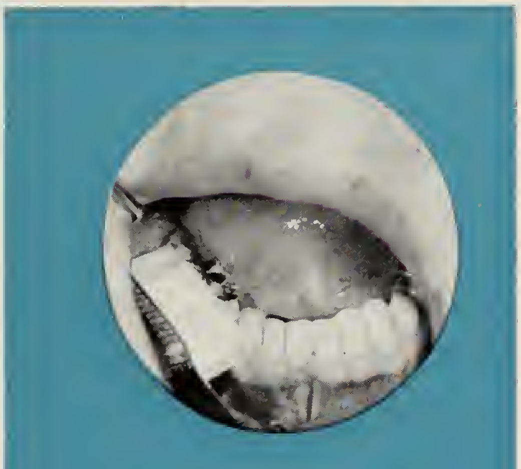


**Fig. 4b**
Position of brush for cleansing the buccal surfaces of the molars and second bicuspid.

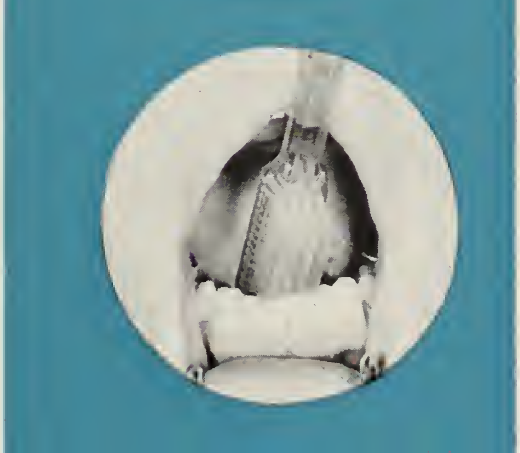


**Fig. 4c**
Vertical position of brush for the lingual surfaces of the lower anterior teeth—used in the same manner for the lingual of the upper anteriors.

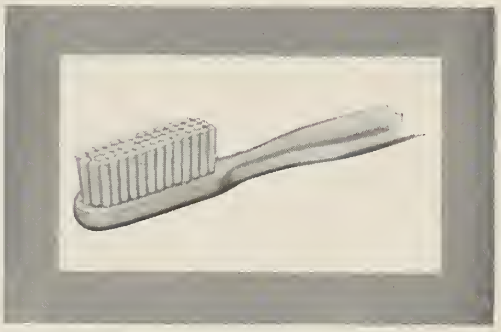


**Fig. 4a**
A four row level trimmed toothbrush.

**OLIN KIRKLAND,**
**D.D.S.**

Associated lecturer on Periodontia, Emory University, School of Dentistry, Atlanta, Ga., Instructor in Surgical Periodontia (Post Graduate) Tufts College School of Dentistry, Boston, Mass., Private Practice, Periodontia, Montgomery, Ala.

PUBLICATIONS: Contributions to Dental Literature, Oral Surgery (Meads). Textbook of Periodontia (Miller). Paradencio Patologia y Tratamiento (Francisco M. Pucci).

HONORS: Past President American Academy of Periodontology, and the Alabama Dental Association. Past member of the Board of Trustees of the A.D.A. Omicron Kappa Upsilon, Fellow International College of Dentists.

## SURGICAL TREATMENT OF PERIODONTAL DISEASE

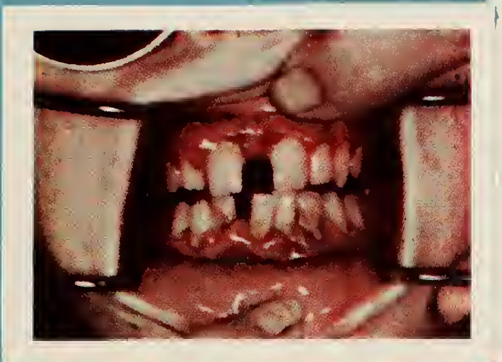
Our leaders in Periodontia, hold many divergent opinions concerning the etiology of periodontal disease. The same lack of unified opinion exists regarding methods of treatment.

Periodontal disease is one of the most common afflictions known to civilization. It is responsible for the loss of more teeth than all other causes combined. It places a burden upon systemic resistance. In most instances periodontal disease can be prevented or cured. Periodontal disease is a dental problem and must be handled by the dentist. In the past, it has been, to a large extent, ignored and neglected by the dental profession.

A better understanding of periodontal disease might result in the prevention of innumerable dental cripples and the salvation of millions of teeth which are annually sacrificed upon the altar of neglect. A more comprehensive understanding of periodontal problems could raise the health standard of our people and perhaps boost the prestige of dentistry to a much higher level. We know some causes of periodontal disease. We also know how it can be eradicated. We, however, have not agreed on a standardized technique which might be used as a pattern for teaching and for daily practice.

Periodontists generally agree that periodontal disease can be successfully treated by surgery.

The progressive development of periodontal disease from the formation of the periodontal pocket to the pathologic exfoliation of the tooth may be checked at any stage of its development. In the early manifestations of the disease, the treatment consists of thorough oral phophylaxis and correct home care of the oral tissues. Where resorption of the bone



and periodontal fibers have progressed to any appreciable extent, a gingivectomy technique is indicated. It should be emphasized, however, that gingivectomy is not a substitute for meticulous scaling of the root surfaces. Its real purpose is to facilitate the application of technical skill by providing the benefits of visibility and accessibility.

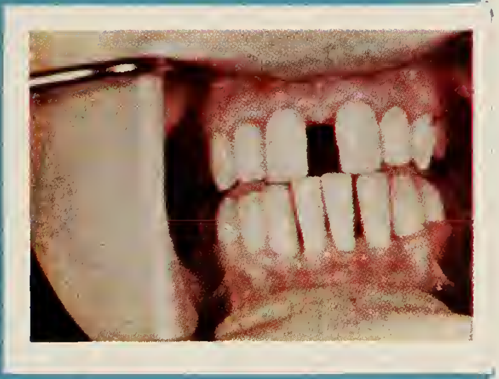
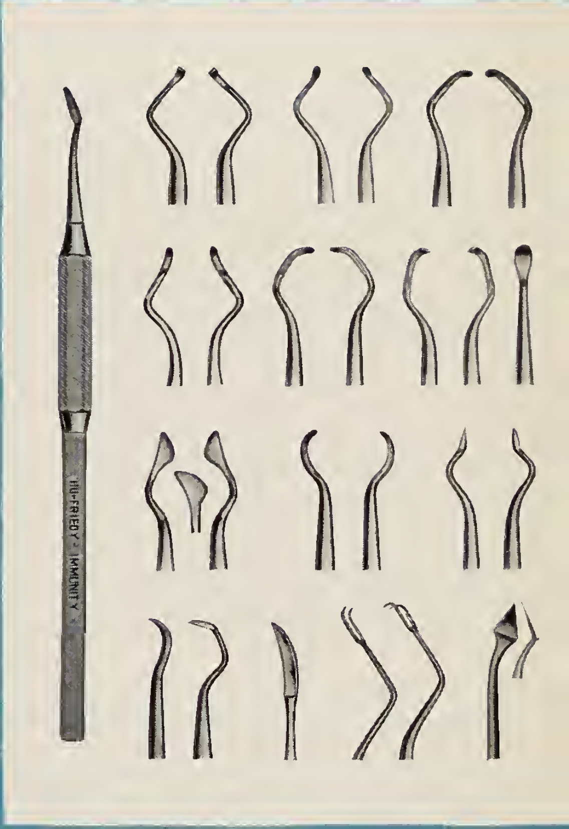


**Fig. 5**
The instruments illustrated comprise the equipment as recommended in the technique defined by Dr. Olin Kirkland.



**OLIN KIRKLAND,**
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# SURGICAL TREATMENT OF PERIO

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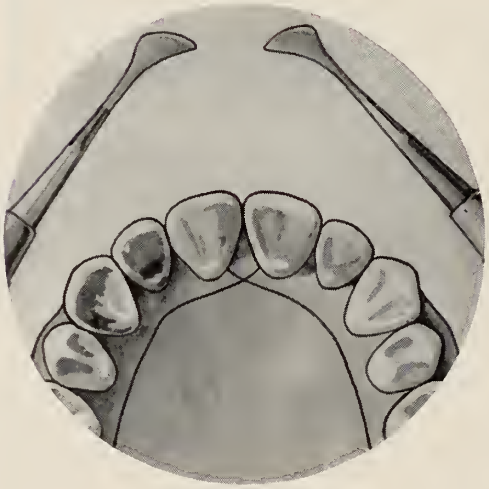

**Fig. 1**
Horizontal line of incision on the palatal surfaces of the teeth.

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**Fig. 2**
Preoperative. Periodontal disease associated with Necrotic Gingivitis.

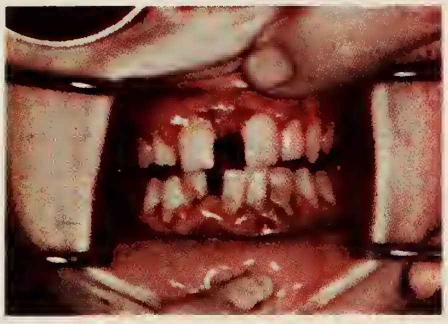
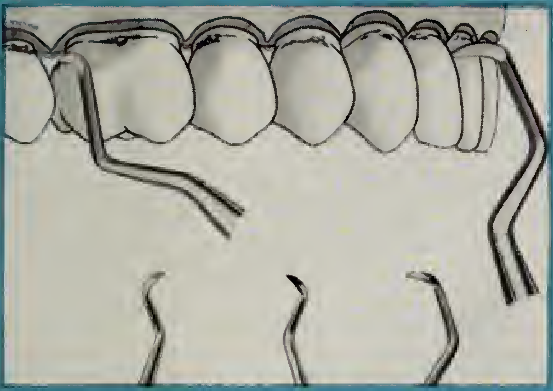


**Fig. 3**
Gingivectomy showing festooning effect on the buccal and labial exposures of the teeth.



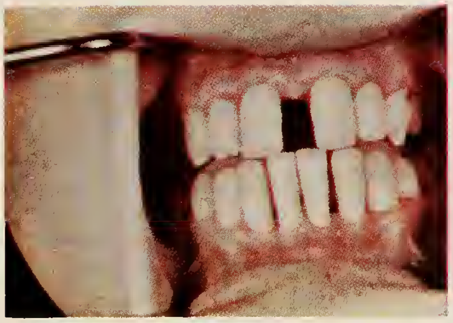
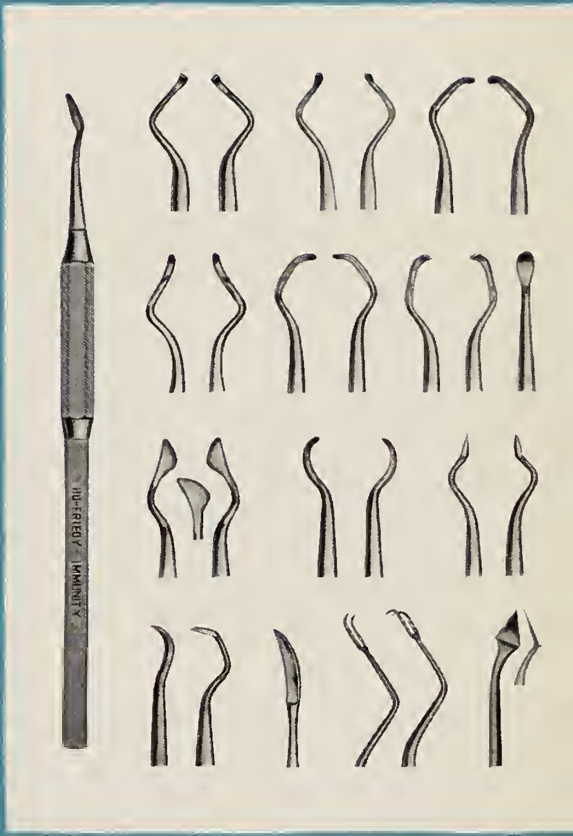


**Fig. 4**
Post-operative two years following gingivectomy.

**Fig. 5**
The instruments illustrated comprise the equipment as recommended in the technique defined by Dr. Olin Kirkland.



**b 5**

PERIODONTIA

**SAMUEL CHARLES MILLER, D.D.S.**

Professor of Periodontia, Chairman of Department of Periodontia, New York University College of Dentistry, New York City.

AUTHOR: Practical Periodontia (with Dr. Sidney Sorrin), 1928. Oral Diagnosis and Treatment (with 28 Contributions), 1936, 1946. Textbook of Periodontia, 1938, 1943, 1950.

HONORS: Fellow: American College of Dentists, American Academy of Dental Medicine, American Association for the Advancement of Science, Associate Fellow: New York Academy of Medicine. Past President: American Academy of Dental Medicine, Omicron Kappa Upsilon (Omega Chapter).

# OCCLUSION AND ITS RELATION TO PERIODONTAL DISEASE

Among the dysfunctional factors which cause periodontal disease, faults in occlusion play the major role. Too little as well as too much stress on the teeth can be involved in the causation of periodontal disease. Individual tooth equilibrium is necessary. This implies a balance between the stresses applied to the individual tooth and the strength of the alveolar support. A case may be in balance in all excursions and still traumatic occlusion exists because the inclined planes are too steep. There is also a relation between cusp height and the age of the patient.

## STEPS IN THE EQUILIBRATION OF OCCLUSION FOR NATURAL DENTURES

### 1. Preliminary Centric

Check centric occlusion and relieve those teeth which are being severely traumatized vertically in this position. Ocular observation during the approximation of the jaws, digital palpation, softened strips of baseplate wax, or articulating paper placed between the contacting surfaces, may be employed to discover premature contacts in centric occlusion. Relieve for food impactions when necessary.

### 2. Protrusive Position

Shape the upper anterior teeth for esthetics. Relieve the lower anterior teeth to the upper anterior line until as many anterior teeth as possible make contact. Posterior teeth should make contact in the protrusive position where possible. Endeavor to avoid producing a loss of contact of anterior teeth in centric position.

### 3. Protrusive Excursion

Produce uninterfered movement from centric position to protrusive position. Relieve for displacement of the upper anteriors as the lower teeth glide along the upper teeth. Grind the upper teeth only, when possible. Relieve for interference of posterior teeth in this movement, being careful not to grind those tooth surfaces that may be required in some other position. Where either upper or lower anterior teeth are being traumatized in this excursion and sufficient relief is not being obtained through grinding of the upper teeth, it is permissible to grind the lower teeth even to the point of taking them out of contact in centric relation, providing the protrusive excursion is deemed sufficiently free to prevent the extrusion of the lower anterior teeth.

### 4. Lateral Excursions

Grind upper teeth from, but not including, the point of centric contact along their planes to produce cusp guidance. Produce free uninterrupted gliding of teeth in the lateral movements. Have as many points of contact as possible on as many teeth as possible but, of even more importance, have free gliding and tooth form commensurate with age. Do either left or right lateral excursion first and then the other. Do not attempt both at a time. Relieve the lingual surfaces of upper anterior teeth where they are found to interfere with lateral movements. As a rule, when grinding posteriors, confine the grinding to the buccal cusps of the upper and the lingual cusps of the lower teeth to avoid reducing the degree of opening.

### 5. Centric Occlusion Check-up

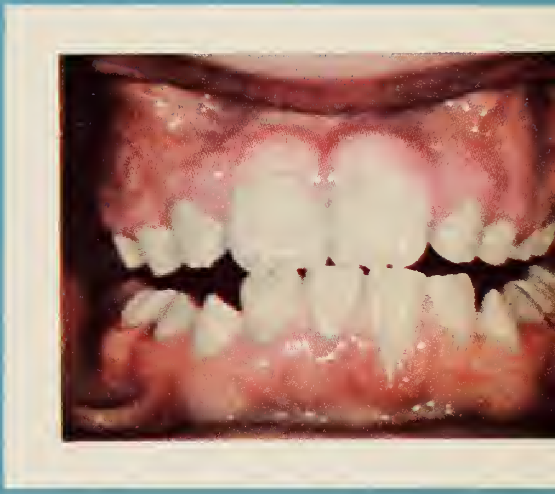
Recheck for simultaneous contact of all teeth in the centric occlusion. Where anterior teeth do not make contact in this position, do not grind the posterior teeth to establish contact of the anterior teeth, as this will result in closing the bite. Ordinarily, very little grinding is necessary at this time. Occlusal sound, the patient's sensitivity, articulating paper, softened baseplate wax and palpation can all be utilized here to advantage as in the first step of balance.

### Summary

Occlusion is fundamental to the practice of dentistry. Occlusal dysfunctions are important factors in the etiology of periodontal disease. The technique of the equilibration of occlusion is simple and can be carried out by the general practitioner. Spot grinding is taboo.

**Fig. 1**

**Effect of traumatic occlusion. Note the gingivitis and recession.**



SAMUEL CHARLES MILLER
D.D.S.

# OCCLUSION AND ITS RELATION

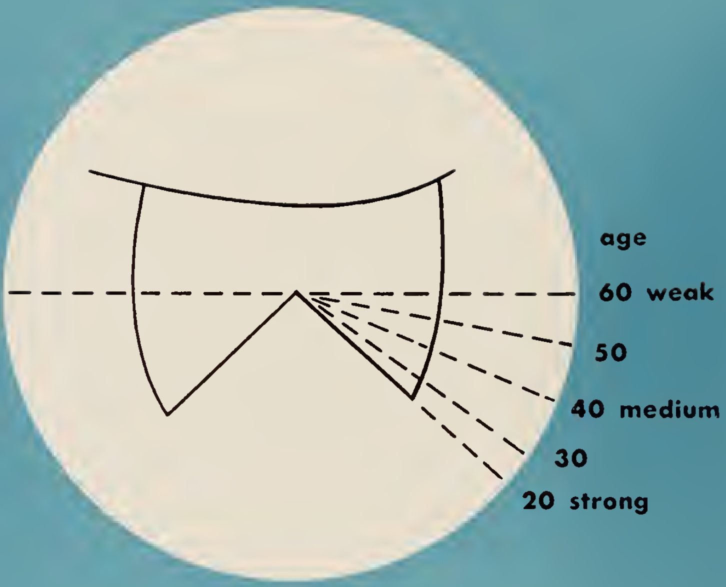


**Fig. 2**
Diagram showing the relationship of cusp height and steepness of inclines to age and alveolar support. (From Miller, Textbook of Periodontia, (Ed. 3) Blakiston, Philadelphia, 1950)

**Fig. 4**
Proper grinding for bicuspids and molars in lateral movements. Possible changes in lateral guiding inclines reducing horizontal stress in lateral eccentric positions. The upper lingual cusps and the lower buccal cusps maintain the maxillomandibular opening and serve as the cutting instrument in the trituration of food. (From Miller, Oral Diagnosis and Treatment, Ed. 2 Blakiston, Philadelphia, 1946)

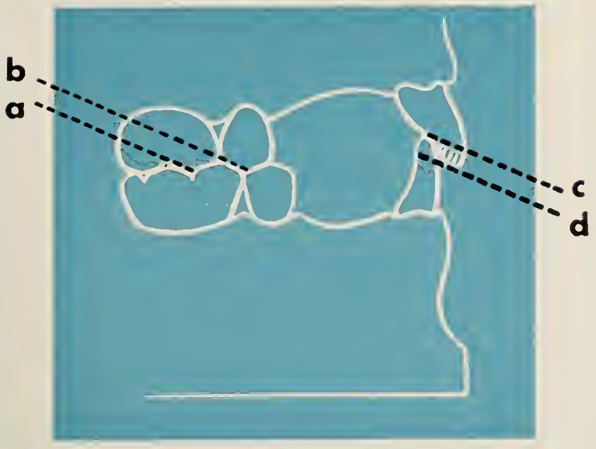
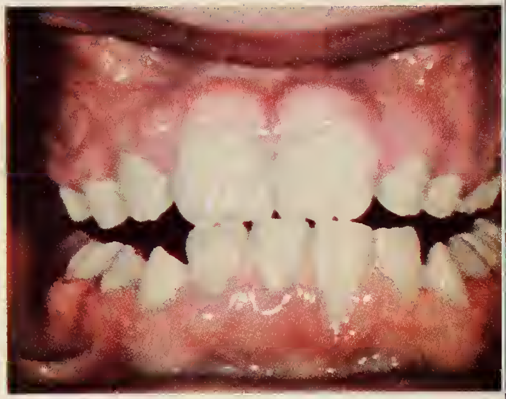


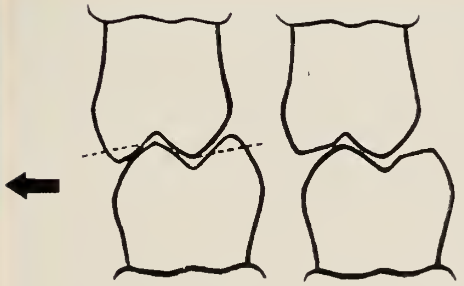
Fig. 3

Relief in the protrusive position. The lateral incisor, cuspid and first pre-molar teeth are not drawn so as to enable the reader to follow the method of grinding. **a** and **b** represent the planes over which the cusp surfaces will glide in a protrusive excursion. These should harmonize with the planes of the anterior teeth. It is obvious that the plane over which the lower central incisor will glide is out of mechanical harmony with the posterior inclined planes. To harmonize the planes, the shaded area of the upper tooth should be removed; thus plane c would haromize with planes **a** and **b.** Contact of the central incisor is then retained in the centric position and harmony in guiding and distribution of stress in protrusive excursion is established. The removal of the area above the dotted line **d** on the lower central incisor would eliminate premature contact in protrusive excursion, but would take the central out of contact in centric and a portion of the protrusive excursion. It may eventually extrude and again traumatize the upper tooth. Change of inclination of the plane of the upper tooth also reduces the stress at the apex of the upper central incisor. (From Miller, Oral Diagnosis and Treatment, (ed. 2) Philadelphia, Blakiston, 1946)

**Fig. 1**

Effect of traumatic occlusion. Note the gingivitis and recession.





**b6**

PERIODONTIA

# NOTES

# QUESTIONS

## NOTES

### CLASSIFICATION AND DIAGNOSIS

1. Does the inflammatory reaction of the gingiva penetrate into the periodontal membrane?
2. Is chronic granulation tissue a diseased tissue?
3. Is alveolar bone diseased in periodontitis?
4. What is the difference between conservative and radical surgery?
5. What is the difference in post-operative care in wounds of primary healing and secondary healing?

### THE PROBLEM OF REATTACHMENT

6. How does one determine clinically the type of pocket?
7. What is meant by "reattachment"? Discuss the various considerations for this problem.
8. What is the epithelial attachment? Discuss its role in the pathogenesis of a periodontal pocket and in the achievement of reattachment.
9. What techniques are employed to attempt reattachment?
10. When does subgingival curettage fail in the treatment of the intrabony pocket?

### SUBGINGIVAL CURETTAGE

11. What are the essentials for thorough subgingival instrumentation?
12. What two factors are of importance in the repair of the periodontal pocket?
13. Are packs ever used in connection with subgingival curettage? For what purpose?
14. How can the general practitioner develop the skill necessary for proper instrumentation?

## NOTES

## QUESTIONS

15. What is the role of the general practitioner in the prevention or treatment of periodontal disease?

### Home Care

16. Should toothbrushes of "hard" bristle texture be recommended?

17. Should patients make a special effort to massage the gingival tissues?

18. What is the procedure for proper care of the interproximal areas?

19. How can a patient be best instructed in proper home care?

20. What is the role of mouth washes in home care?

### SURGICAL TREATMENT

21. Is it possible to prevent or cure periodontal disease?

22. What advantage has gingivectomy over other methods?

23. Why should the labial and buccal gingivae be removed with festooning effect?

24. Why is it unnecessary to use festooned incisions on the lingual exposures of the teeth?

25. What is the importance of post-operative treatment?

### OCCLUSION

26. What causes traumatic occlusion?

27. Define: (a) Centric occlusion (b) Centric relation (c) Physiologic rest position.

28. What is bruxism and how does it affect the periodontal structures?

29. What is individual tooth equilibrium?

30. What is meant by food impaction? How may it be relieved?

PERIODONTIA

# NOTES

**STANLEY D. TYLMAN, D.D.S. M.S.**

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PUBLICATIONS: Research in metallurgy and dental materials; resilient resins; somatoprostheses; cleft palate prostheses and complete oral rehabilitation.

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# CLINICAL AND PHYSICAL APPRAISAL OF DENTAL MATERIALS

**Monday, January 8, 1951**

## OUTLINE

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| 5. Silicate Cement and Amalgam Fillings | G. Paffenbarger |
| 6. Gold Inlay and Gold Foil Restorations | G. M. Hollenback |

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## SUMMARY

**THE REASON FOR A BIOLOGICAL AND PHYSICAL APPRAISAL.** A tooth impaired in its function is either restored with a filling or, if beyond repair, it is extracted; if extracted it is replaced with a bridge. In either case the filling or the bridge is a therapeutic means for correcting or restoring impaired or lost physiologic functions.

Restorative dentistry in its broader sense implies an understanding of the biological structures and functions of the dental tissues; both the hard and the soft tissues. It also requires a knowledge of the physical properties of the materials used in constructing fillings or bridges. Finally, a comprehension of the forces exerted upon these restorations and their reactions upon the dental tissues is also essential.

Our knowledge of the histological structure of dental tissues has increased notably during the past two decades. Along with this has come a fuller grasp of their biologic processes. Concurrently there has been an increased interest in the physical phenomena of dental materials used in dentistry, with the result that acceptable materials meet definite specifications formulated by the United States Bureau of Standards. These specifications insure desirable chemical and physical qualities in the materials offered to the profession by manufacturers.

Many problems and failures of the past have been resolved through a better understanding of the physical phenomena which accompany the use of filling materials.

Discomfort, pain, even the death of the pulp, can be directly attributed to improper manipulation and application of filling materials. The dislodgement of fillings, fracture of teeth and other mishaps may be explained by the disregard of fundamental mechanical and physical laws.

Each new filling material requires a complete appraisal of its physical properties first in the testing laboratory, then in the mouth to check its biological compatibility with the living tissues. Thus physical and mechanical laws are correlated with biological reactions.

Although amalgam is one of the oldest filling materials used in dentistry, there are still many unknown factors related to its use. The cast gold inlay is far from being the perfect filling even with the great advances that have been made in improving the waxes, casting investments, gold alloys and methods of precision casting.

The silicate cements leave much to be desired and the new self-curing resins are in their developmental stages. Many of their physical phenomena are baffling and their biological reactions unknown.

The scope of this discussion of the biological factors must of necessity be limited, likewise the consideration of the physical and mechanical laws governing the use of the dental materials.

The biologic aspects are considered first, since they form the broad base upon which all restorative dentistry rests. The mechanical principles which govern the preparation of teeth to receive fillings are equally important; they span the gap between the biological and the physical phases. In logical sequence our discussion centers around the five types of filling materials most generally used today; namely: amalgam, silicate cements, acrylic resins, cast gold inlays and gold foils.

A chart has been prepared wherein the various dental materials are appraised on the basis of their clinical, biological and physical factors. (See supplementary chart on Appraisal of Dental Materials). A quick survey of each material will give not only its clinical indications and contra-indications, but also its relative position in the scale of desirable or undesirable physical properties.

# C

## APPRAISAL OF DENTAL MATERIALS

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# THE BIOLOGIC ASPECTS

At the beginning of the century, most dental clinicians regarded the tooth as an inanimate object. Operative procedures were examined only from their mechanical aspects. Filling materials were examined only for their physical and aesthetic properties. As dental science progressed it became apparent that operative procedures and filling materials, as well as caries, produce definite effects upon the dentin and the pulp.

More recently histologic studies have shown that filling materials can have a profound effect on the dentin and pulp (Figure 1). Some filling materials, like zinc oxide and eugenol paste, protect the pulp and stimulate it to a defensive response. Other materials, like silicate cements, may irritate the pulp and lead to permanent injury.

Today filling materials must meet *three* basic requirements:

1. Physical
2. Aesthetic
3. Biologic

The biologic requirements are assessed on the basis of the effects of these materials upon the histologic structure and function of the dental tissues. These effects may range from a desirable sedation of the pulp and *stimulation* of secondary dentin formation to an undesirable *irritation* which may eventually lead to the death of the pulp.

## BIOLOGY OF THE DENTAL STRUCTURES

**Enamel.** Enamel is the hardest structure in the body. It is acellular and, therefore, non-vital. It is incapable of self-repair. Enamel, however, does conduct pain-producing thermal changes caused by extreme cold or heat coming in contact with the tooth surface. It also conducts to the pulp heat generated during operative procedures.

The high degree of mineralization of the enamel makes it brittle. Fillings should therefore be placed so that they are supported by enamel rods which rest on sound dentin lest the enamel margins break. Enamel is also soluble in acids. Filling materials of acid reaction should, therefore, be avoided lest the enamel walls undergo some decalcification.

**Dentin.** Dentin is also calcified and avascular, but differs from the enamel in that it is 40 per cent organic. It is, therefore slightly elastic and does not fracture easily. The dentinal tubules contain the processes of the odontoblasts in the pulp. These tubules are the living vanguard of the cells in the pulp and confer vital reactivity to the dentin. Depending on the nature of the stimulus, the dentin reacts favorably or unfavorably. The favorable or *defensive response* consists of *a*, hypercalcification of the dentinal tubules (sclerotic or transparent dentin); and *b*, formation of secondary dentin in the pulp. The *unfavorable response* consists of a degeneration of the odontoblastic processes and of the pulp (Figure 1).

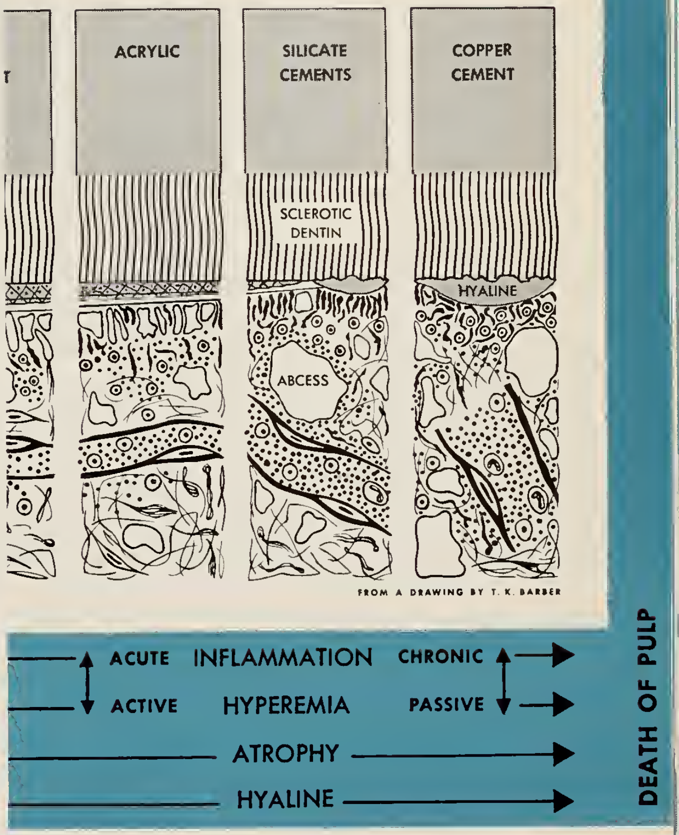
**Pulp.** The pulp is a richly vascular and loose connective tissue. It is highly vital and sensitive. It readily responds to rapid and extensive cutting of the dentin, exposure to saliva, irritant filling materials or caustic drugs. As a result of these irritants hyperemia may set in and lead to self-strangulation of the pulp because of the confining walls of the pulp and the constriction at the apical foramen. In that event, formation of secondary dentin does not occur.

**Gingiva.** The epithelium of the gingival sulcus is not keratinized. It is therefore highly sensitive to mechanical, chemical and bacterial irritations. Thus, any filling which is rough, porous, or has an overhanging margin leads to gingival irritation and inflammation. Fortunately, the gingiva usually has high regenerative power. The effect of fillings upon the gingiva is a neglected field in dental research.

**Periodontal Membrane.** The periodontal membrane consists of a rich supply of collagenous fibers arranged to attach the root to the olveolar bone. The direction of the fibers is also designed to sustain and distribute the forces of mastication. Many of the fibers follow an oblique course so that the tooth is best equipped to resist forces in the axial plane.

A "high" filling which places the tooth in surpa-occlusion may result in the crushing and injury of the periodontal membrane.

Injury of the membrane may also be caused by the application of excessive and lateral forces during the insertion of a gold foil restoration.



FROM A DRAWING BY T. K. BARBER

he effects vary in quality and degree with many factors;

1. distance of the filling material from the pulp
2. proximity of the pulp
3. extent and depth of cavity
4. rapidity of cutting dentin
5. heat generated during cavity preparation (hand instruments, burs, abrasives)
6. presence and duration or absence of caries and attrition
7. presence of secondary dentin
8. age of patient
9. time elapsed since the insertion of the filling
10. individual reaction of the pulp

ISAAC SCHOU
D.D.S., Ph.D., D.S

# THE BIOLOGIC ASPECTS

## BIOLOGIC REACTIONS TO DENTAL MATERIALS

Figure 1 is a schematic and diagrammatic representation of the response of the pulp to various filling materials. Our present state of knowledge is insufficient to give an accurate and conclusive summary of all the effects which may be observed under each type of filling. However, sufficient facts are known to point out that operative procedures and filling materials do influence the odontoblasts, the connective tissue cells, the vascular supply of the pulp and the formation of secondary dentin.

The pulpal reaction will vary in character and severity according to many factors (see Footnote to Figure 1). These factors will determine whether the resulting pulpal changes are

a) favorable;
b) unfavorable but temporary and still reversible; or
c) unfavorable and irreversible, therefore, resulting in the death of the pulp.

Operative procedures and dental materials are inseparable. Poor operative procedure will offset desirable qualities of dental materials. Poor dental materials will offset proper operative procedure.

**Zinc oxide-Eugenol Cement.** Investigators, (Zander, Manley, and Gurley and Van Huysen,) are generally agreed that eugenol cements produce the least pulpal reaction of all filling materials (Figure 2). It should be used whenever possible as a base to alleviate unfavorable pulpal reactions induced by caries or operative procedures. This is especially indicated in young patients. The antiseptic and obtundent action of the eugenol may be responsible for its effectiveness.

**Gutta Percha.** This material is chemically inert and is non-irritating if placed with care and without undue pressure upon the cut dentin.

**Amalgam.** Amalgam may lead to a decrease in the number of odontoblasts, hyperemia, and round cell infiltration in the pulp. These effects may be the result of thermal and possibly galvanic action, and are aggravated when the cavity is deep and the amalgam is close to the pulp without an intervening cement base.

**Zinc Cement.** The zinc oxyphosphate cements cause a more severe effect upon the pulp than does amalgam. This might be due to their exothermic and acid action.

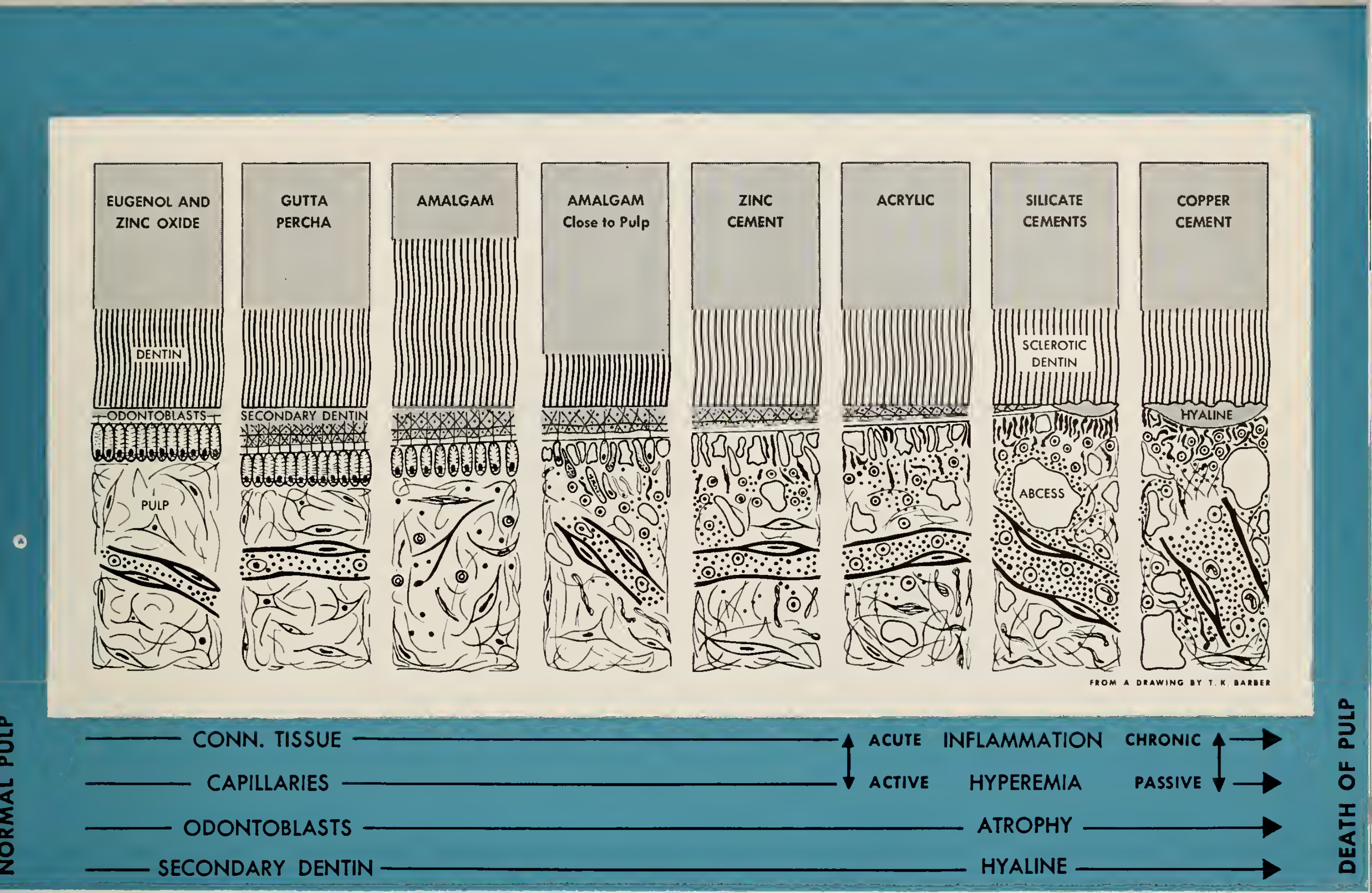
**Acrylic Fillings.** Self-curing acrylic restorations may be harmful to the pulp and lead to cellular degeneration, hyperemia, and round cell infiltration or fibrosis. An impermeable lining should be used to give the pulp some protection. However, further testing of this material is needed.

**Silicate Cement.** Silicate fillings sometimes lead to pulpal inflammation, degeneration of the odontoblasts, severe hyperemia, and finally abscess formation and necrosis (Figure 3). The mechanism of such injury is not established. The chemical agents in the silicate, the acid used, or the poor sealing quality of the material may be the cause of the injury.

Zander and Pejko, (Jour. A.D.A. June, 1947), point out the advisability of first placing a non-irritant temporary filling in order to allow the pulp to form a protective layer of secondary dentin before the final restoration of silicate cement is inserted.

**Copper Cement.** Published reports indicate more severe pulpal reactions following the placement of copper cements than with other filling materials. Formation of hyaline instead of secondary dentin and pulpal abscesses have been described.

# PULPAL RESPONSE TO FILLING MATERIALS



FROM A DRAWING BY T. K. BARBER

**Fig. 1**

Footnote: The effects vary in quality and degree with many factors;

1. distance of the filling material from the pulp
2. proximity of the pulp
3. extent and depth of cavity
4. rapidity of cutting dentin
5. heat generated during cavity preparation (hand instruments, burs, abrasives)
6. presence and duration or absence of caries and attrition
7. presence of secondary dentin
8. age of patient
9. time elapsed since the insertion of the filling
10. individual reaction of the pulp

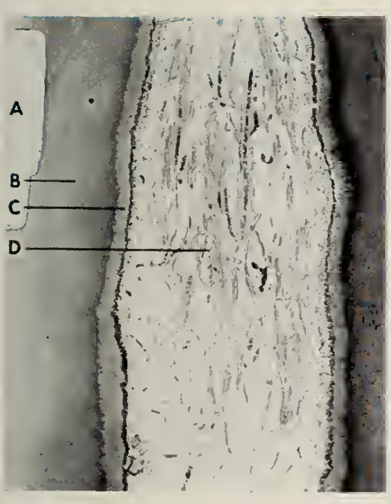


**Fig. 2**

Normal Pulp under Zinc Oxide-eugenol Cement.

Lower right lateral incisor of patient. (A) cavity; (B) dentin; (C) uncalcified dentin; (D) pulp. This was a sound tooth filled with zinc oxide-eugenol cement for six weeks before extraction. The pulp is normal. (Courtesy of Zander, Jour. Am. Dent. Assn. June 1947)

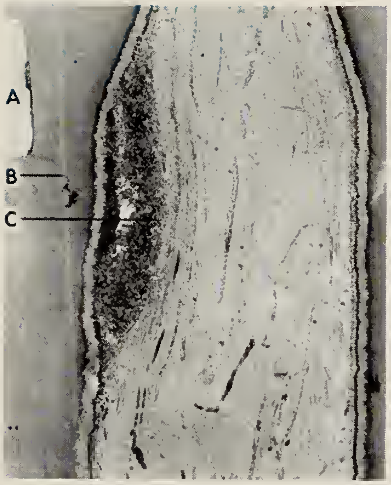


**Fig. 3**

Severe Pulpal Changes Following Insertion of Silicate Cement.

Lower left cuspid of patient. (A) cavity; (B) dentin; (C) pulp inflammation. This was a sound tooth filled with silicate cement for six weeks before extraction. Inflammatory reaction is present adjacent to the dentinal tubules leading to cavity floor and wall. (Courtesy of Zander, Jour. Am. Dent. Assn. June 1947)

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APPRAISAL OF

DENTAL MATERIALS

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# THE BIO-PHYSICAL PRINCIPLES OF CAVITY PREPARATION

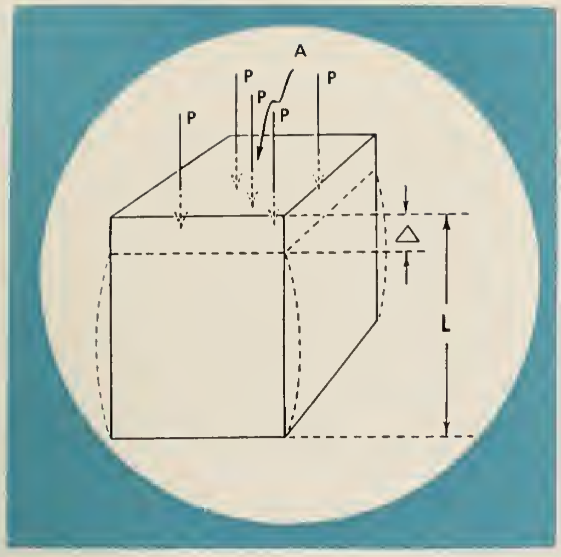
The masticatory apparatus is a machine whose function is the utilization of the force of muscle contraction for the comminution of food. This tensile force is transmitted and modified by means of a lever, the mandible, and is applied to the food as compressive and shearing force by means of the inclined planes of the opposing teeth of both jaws.

The parts of a machine, whether of inanimate or of vital substance, undergo a slight change of shape under the applied force, known as strain. The force set up within the materials of a machine as a result of the strain opposes the external force producing the strain and is known as stress.

When a machine is functioning, several things may happen. If it is loaded too heavily, i.e. the applied force is too great, or some of its parts are weakened, the strain may be so great that the parts will be broken or permanently deformed. When, however, it is functioning properly, the strains and stresses are proportional to the forces acting on the parts and are zero when the machine is at rest.

Since different parts of the machine have different dimensions as well as different loads, it is necessary to reduce the stresses and strains to those in volumes of unit dimensions. It is, therefore, customary to speak of pounds per square inch (psi) (Fig. 1) and inches per inch respectively.

As the parts of a machine transmit force and the mechanical properties of materials are expressed in the same term, it is more convenient to speak of stress rather than strain. But this makes no difference since within useful limits the ratio of stress to strain (Fig. 1) is a constant quantity for a given material. This quantity is known as the modulus of elasticity.



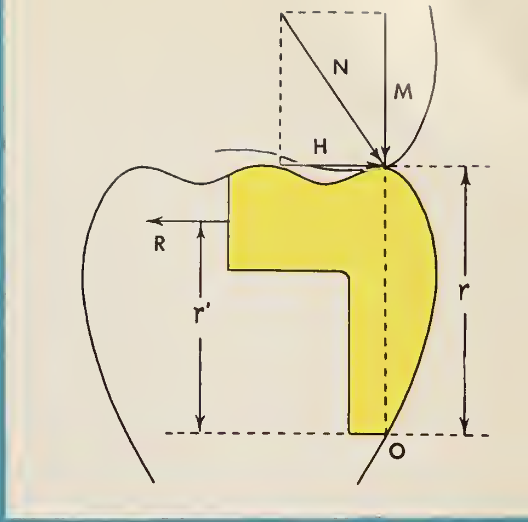
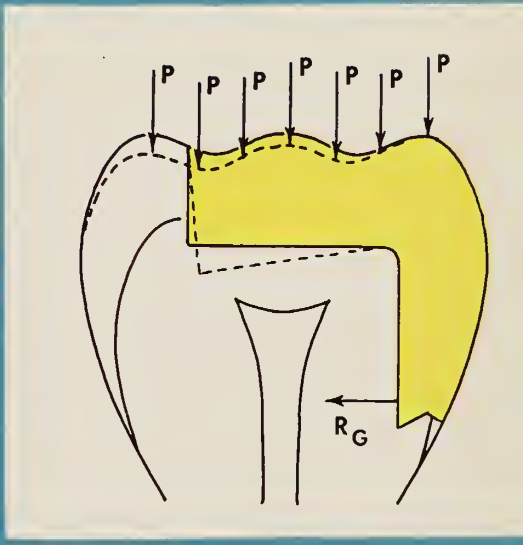


**Fig. 2**
Section of tooth with proximocclusal restoration and opposing cusp making contact on marginal ridge only. The arrow N represents the reaction of the cusp, arrow H its horizontal component and arrow M its vertical component which is equal and opposite to the force of mastication. Also R:H = r:r′.



The teeth are the essential parts of the masticatory machine in the sense that the drill is the essential part of the drill press. The stresses set up in them may be very large. Sound enamel and dentin are strong and amply able to withstand these stresses, but when the tooth is weakened by decay and restored by one of the materials in use today, the whole mechanical structure of the tooth is changed. For instance, the unsupported buccal and lingual cusps of an extensive proximocclusoproximal cavity in a bicuspid now become vertical cantilever beams and may be broken off at the level of the gingival floor by the wedging action of an opposing cusp. This may be prevented by covering the cusps so that the loading is axial instead of transverse. Or again, the proximocclusal restoration requires an occlusal lock (Fig. 2) to retain the restoration against the wedging force of an opposing cusp. This makes contact with the natural cusps of the restored tooth and the marginal ridge of the restoration since there is no cohesion between the tooth and the restorative material.

The most common difficulty is probably that of obtaining adequate retention. A mechanical lock is recognized as the only satisfactory type of retention. Its placement, however, is not always a simple matter, particularly where much tooth structure has been lost. The lock should be placed so that its axis is parallel, or as nearly so as possible, to the horizontal component of force. This will prevent bending in a horizontal plane and avoid greatly increasing the stresses in parts of the tooth and the restoration.

Another problem is presented by the mechanical lock on the occlusal surfaces of molars and bicuspids, where the modulus of elasticity of the material is considerably greater than

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## THE BIO-PHYSICAL PRINCIPLES

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that of the dentin. This is the case with hard inlay golds in all teeth and probably explains some of the failures of amalgam in deciduous molars due to their softer and less dense dentin. The dentin being more compressible than the gold, the remaining occlusal surface of the tooth, including the occlusal lock, tends to be deflected more under the vertical force than the proximal portion of the restoration. This may result in rotation inward of the occlusal lock and outward of the gingival portion about an axis in proximity to the axiopulpal line angle. Fracture of the restoration at this angle due to bending (Fig. 3) may also occur if the restoration is locked at the gingival floor.

Many of the problems that arise in dental practice can be correctly solved only by the application of well known mechanical principles. These problems are, however, made more difficult by biological considerations such as the necessity for putting pulp preservation above mechanical considerations and the need to conform to the external anatomy of the tooth.

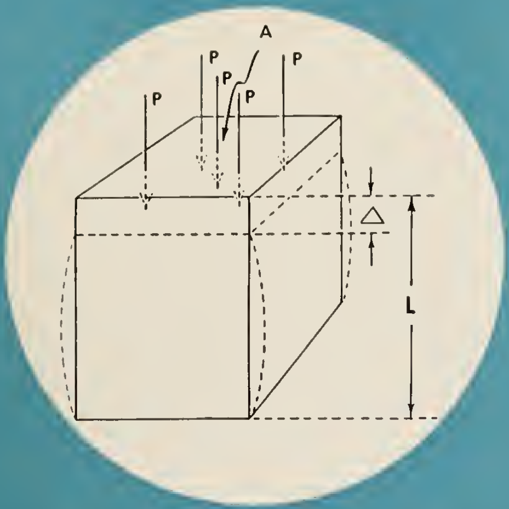

**Fig. 1**

Prism subjected to axial loading. The total load P equals the sum of the p's. The unit stress equals the load P divided by the area A, or $S = P/A$. The unit strain $\sigma$ equals the total strain $\triangle$ divided by the length L, or $\sigma = \triangle/L$. The unit stress S divided by the unit strain $\sigma$ is a constant quantity up to the proportional (elastic) limit. This important quantity is known as the modulus of elasticity E, or $E = S/\sigma$.

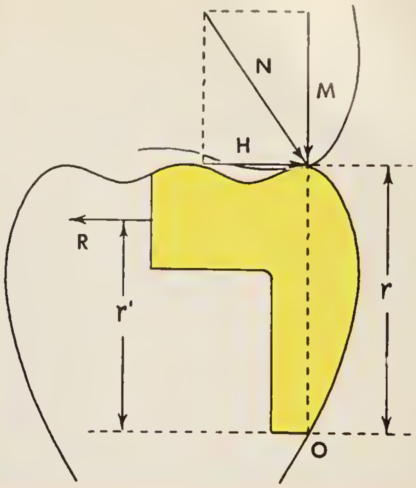

**Fig. 2**

Section of tooth with proximocclusal restoration and opposing cusp making contact on marginal ridge only. The arrow N represents the reaction of the cusp, arrow H its horizontal component and arrow M its vertical component which is equal and opposite to the force of mastication. Also $R{:}H = r{:}r'$.

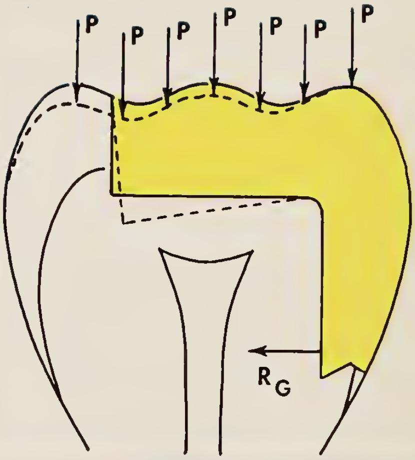

**Fig. 3**

Section of tooth with proximocclusal inlay with gingival retention, under a uniformly distributed load, showing compression of tooth and bending of inlay at axiopulpal line angle.

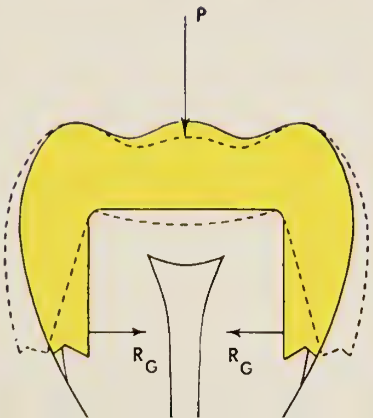

**Fig. 4**

ection of tooth with
OD inlay showing
ending under a con-
ntrated load and ro-
tion of gingival por-
ons without locks and
tention with locks.

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APPRAISAL OF

DENTAL MATERIALS

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## SYNTHETIC RESIN FILLINGS

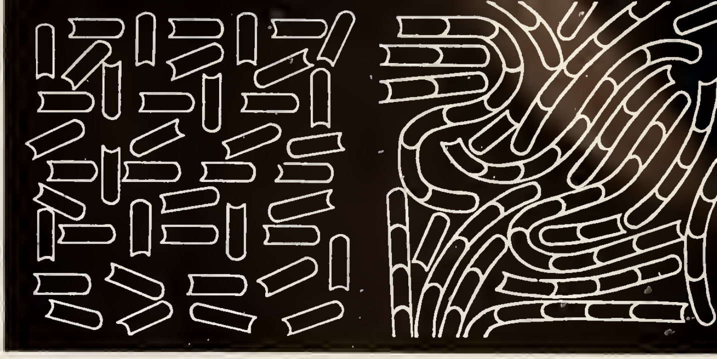
The synthetic resins have been employed as tooth restorative materials since about 1940. During these ten years the dental profession has learned much about the characteristics and behavior of the resins designed for this purpose. They have been recommended by some for the construction of extensive bridges, anterior crowns to replace porcelain, all types of inlays to replace gold, amalgam, or silicate cements, and numerous other extensive types of restorations. Others in the profession have been more conservative in the application of the resins for restorations.

Much has been learned from research studies about the characteristic properties of resins used for tooth restorative purposes. Since most of the products have been either methyl methacrylate resin or a closely related acrylic resin, the chemical nature and general properties of the dental resins are quite well established. When using the indirect resin restoration, which is processed or cured in a mold with the application of heat, it has been found that the processing practice is important in modifying the properties of the finished structure. As a result, the generally accepted practice is to cure the resin mass at a relatively low temperature, around 165° F. until complete curing is assured. The process of curing the resin mass is thought to be one of converting the residual liquid to a solid state by the process known as polymerization. During the polymerization the single molecules of liquid combine with adjacent molecules to form a giant molecule. When this process occurs, some heat is given off from the resin mass, which may modify the properties of the finished structure.

Many of the characteristics of tooth restorative resins have been established. It is recognized that they possess pleasant esthetic qualities of color, taste and cleanliness. When completely polymerized the resins are compatible with the oral tissues. The strength and dimensional stability of the resins have been determined both in service and by physical measurement. In these qualities the resins are not superior. The resins are also less favorable than some other materials in relative hardness, resistance to flow, elastic qualities and resistance to wear.

During recent months resin products of the powder-liquid type have been introduced as direct filling materials which cure spontaneously without the application of heat to the resin mass. Fundamentally these resins are similar to the older products in both composition and properties. The chief difference is that a small amount of an activating chemical is added to cause the curing process to proceed at low temperatures.

While the extent is not yet determined, to which the self curing resins may be employed, it seems practical to consider these materials as possessing possibilities for service in limited types of restorations. When used conservatively the direct resin restoration may serve as a practical material. It is doubtful, however, if these materials should be used where the restoration is subjected to the full force of occlusion.



**Figs. 1-2**
Sketch representing the combination of monomer molecules to form the polymer chain.

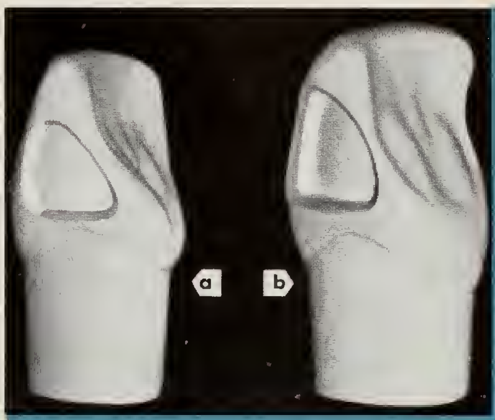


**Fig. 3 (top)**
(a) Typical cavity preparation for reception of acrylic inlay.
(b) Same type of cavity preparation in (a) may be extended.
(Courtesy E. B. Clark: Proc. Dent. Centennial Celebration, 1940).

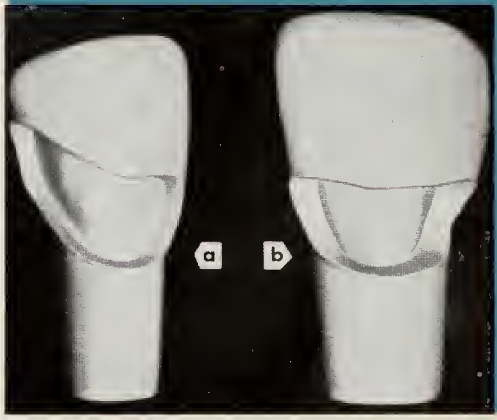


**Fig. 4**
Another type of cavity suited for acrylic-resin restoration. (a) Basic form of such preparation. (b) Where tooth has had previous silicate restoration. (Courtesy E. B. Clark: Proc. Dent. Centennial Celebration, 1940).

## CHARACTERISTIC PROPERTIES OF TOOTH RESTORATION RESINS

(PROCESSED IN BOILING WATER FOR 60 MINUTES)

| PRODUCT | PER CENT LINEAR SHRINKAGE | PER CENT SOLUBILITY* | WATER SORPTION* | BRINELL HARDNESS NUMBER |
|---|---|---|---|---|
| RESIN #1 | 0.35 | 0.10 | 0.59 | — |
| RESIN #2 | 0.36 | 0.07 | 0.45 | 27 |
| RESIN #3 | — | 0.04 | 0.53 | 29 |
| 22-k DARK ALLOY, LAST | — | — | — | 34 |
| AMALGAM | — | — | — | 80-100 |

* AFTER 7 DAYS IN DISTILLED WATER

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# SILICATE CEMENT AND AMALGAM FILLINGS

## SILICATE CEMENT

The powder is essentially a glass, a complex aluminosilicate containing calcium, sodium, fluorides and phosphates. The liquid is an aqueous solution of phosphoric acid to which have been added zinc salts and usually aluminum salts.

On mixing, the liquid attacks the surface of the powder particles to form a gel-like matrix or bonding substance as shown in Figure 1. This gel-like matrix is responsible for the solubility, the staining, the weakness and the irritant properties of silicate cement. Therefore, the technic of mixing and inserting a silicate cement restoration should be designed to hold the amount of the gel-like matrix in a cement to the lowest possible minimum. To do this the cement should be mixed quickly on a chilled slab to a heavy putty like consistency. Such a prepared mix has only one half the shrinkage, is one third less soluble and is about one fifth stronger than a mix made slowly on a warm slab.

These effects are not only easily demonstrated in the research laboratory but are grossly reflected in actual restorations as shown in Figure 2. These two proximal silicate cement restorations were made with the same cement. In "A" the cement was mixed in 30 seconds on a cool slab to a heavy putty-like consistency so the 0.4 g of powder was incorporated into 0.1 ml of liquid. In "B" the same cement was mixed for two minutes on a warm slab to the same heavy putty-like consistency but only 0.3 g of powder could be incorporated into 0.1 ml of liquid because of the rapid rate of reaction between the powder and liquid on the warm slab. Then too restoration "B" was finished immediately while "A" was not dressed down until five days later and then only a small amount, as the excess cement was kept to a minimum by carefully judging the quantity necessary to fill the cavity.

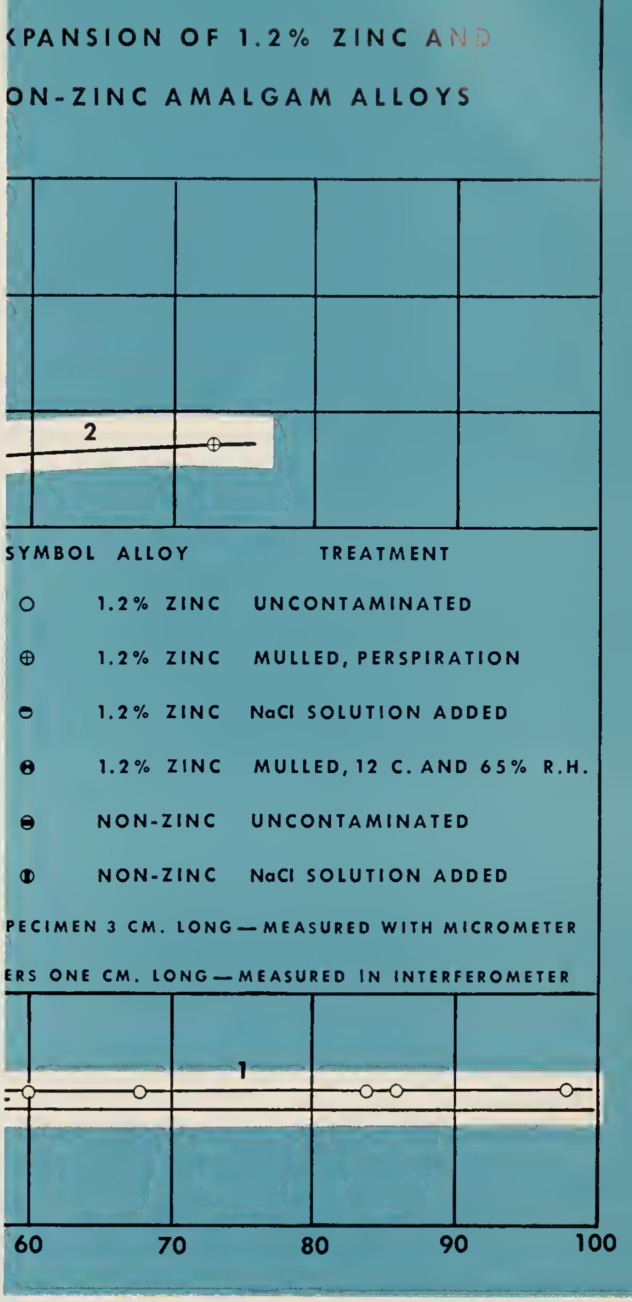
### AMALGAM

Many dentists have noticed that in the same patient even when using the same brand of alloy, some amalgam restorations caused post-operative pain while others did not. Some restorations corroded, blistered or pitted while others retained their polish and margins. Some restorations expanded so much that they actually extruded from the cavity in much the same manner as "4" in Figure 3 while other restorations made in the same mouth, by the same operator and with the same alloy, did not.

It has been only within the last few years that the cause of this seemingly contradictory behavior of amalgam has been explained—the incorporation of moisture during the mixing and packing. Moisture from any source—perspiration, saliva, washing solution, or condensation, if incorporated in the amalgam during mixing or condensing causes corrosion, after-pain, and excessive expansion. The moisture contaminated amalgams must contain zinc, otherwise the excessive expansion and corrosion are not present. Amalgams containing zinc furnish a host of corrosion cells. The moisture present is dissociated into hydrogen and oxygen by the electrical current generated by these cells. The released hydrogen collects and causes the amalgam to swell and to blister on the surface. The oxygen probably reacts with the alloy components to form corrosion products. The presence of salt in perspiration accelerates the reaction. But neither salt nor zinc cause the expansion unless moisture is present. The effect of zinc and the magnitude of the expansion on zinc and zinc free alloys are shown in Figure 4.

### SELECTION AND USE OF SILICATE CEMENTS AND AMALGAMS.

The Federal Government (National Bureau of Standards) and the American Dental Association (Council on Dental Research), in a cooperative research on dental materials, formulated specifications for silicate cement and amalgam. They established a List of Certified Silicate Cements and Amalgam Alloys which comply with those specifications. These Lists of Certified Dental Materials are published from time to time in the Journal of the American Dental Association as a guide for the dentist and his assistants in procuring dental materials. The selection of one of these materials does by no means guarantee a satisfactory restoration unless a proper technic is used. Probably the greatest improvement in current restorative dental service will come with improvement of technics in using both the old and newly developed materials.



algam. J. A. D. A., 29: 1825, October, 1942).

ining amalgam plugs
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tively dry but un-
was not touched by
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4 was palmed in a
and dried with alco-
detected on Nos. 2
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sion on No. 3.

**GEORGE C. PAFFENBARGER**
**D.D.S., D.Sc.**

# SILICATE CEMENT AND AMALG

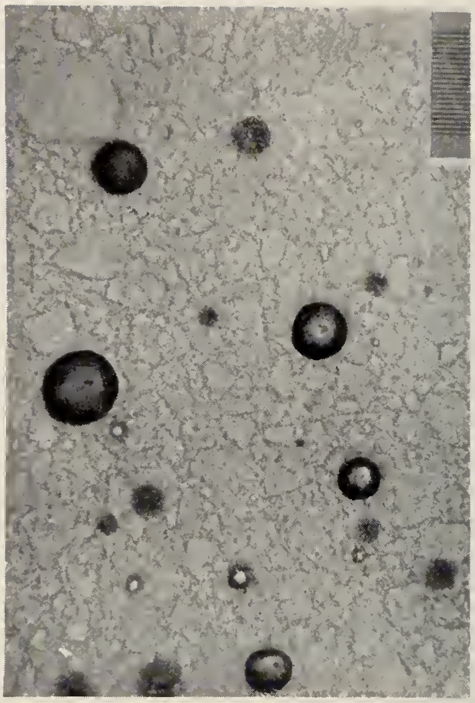

**Fig. 1**
Photomicrograph of a set silicate cement showing air bubbles and powder particles embedded in a gel-like cementing substance or matrix. The glass-like powder particles do not dissolve in the liquid during mixing. Only the outside surface is attacked by the liquid. (Paffenbarger, Schoonover and Souder, Dental Silicate Cements: Physical and Chemical Properties, J. A. D. A., 25:32, January, 1938).

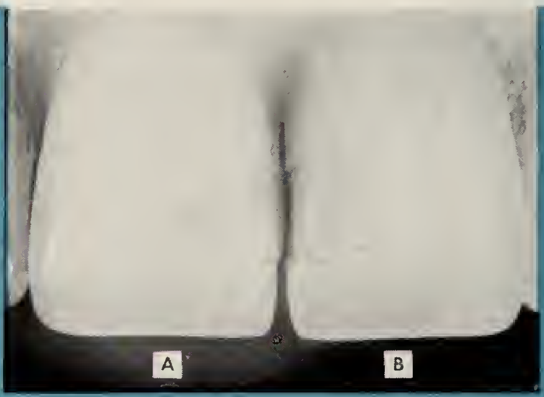

**Fig. 2**
The same brand of silicate cement was used in making these two adjacent restorations. Restoration A was made from a mix spatulated on a slab at 60° F for 30 seconds. Petrolatum was placed on the restoration after removing the matrix which was allowed to remain undisturbed. Restoration B was made from a mix spatulated for two minutes on a slab at 98° F. Immediately after removing the matrix strip, moisture came in contact with the restoration. After one and one-half years the dissolution of restoration B progressed at a far faster rate than did restoration A.

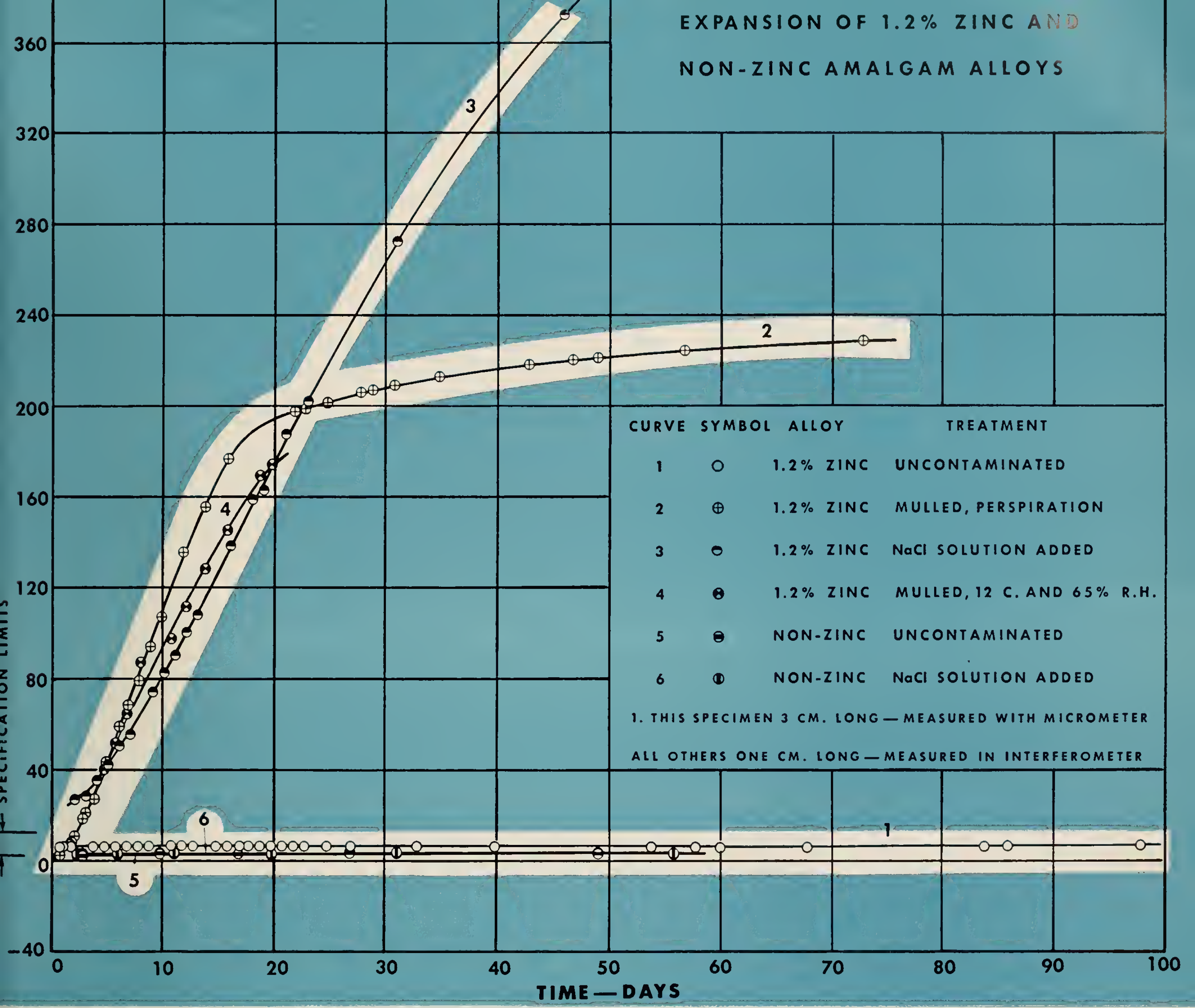

**Fig. 4**

(Schoonover, Souder and Beall. Excessive Expansion of Dental Amalgam. J. A. D. A., *29:* 1825, October, 1942).

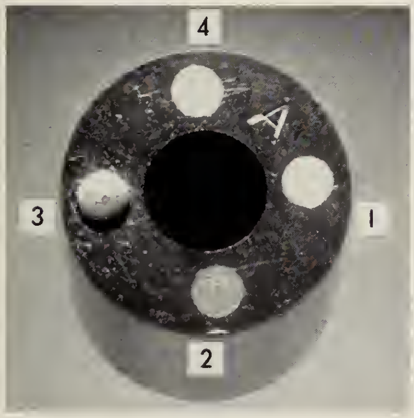

**Fig. 3**

Expansion of zinc-containing amalgam plugs placed in a steel die. Amalgam 1 was palmed in a comparatively dry but unwashed hand; No. 2 was not touched by hand; No. 3 was palmed in a hand wet with salt solution; amalgam 4 was palmed in a hand previously washed and dried with alcohol. No expansion was detected on Nos. 2 and 4. There was a slight expansion on No. 1, and a very large expansion on No. 3.

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# GOLD INLAY AND GOLD FOIL RESTORATIONS

In restoring carious teeth, two broad classifications of materials are in general use.

1. Those materials such as gold foil, amalgam, cement and some plastics. In the use of these materials the restoration is built in direct contact with the walls of the cavity. It depends on close mechanical adaptation of the material to the cavity walls in order to prevent the ingress of harmful foreign matter, such as fluids, bacteria, etc.

2. Those materials such as cast gold, fused porcelain and some plastics. These are preformed outside of the prepared cavity and upon insertion, depend on a cementing medium to produce a hermetic seal, and also to retain the restoration.

There is not complete agreement as to which type of restoration is the most desirable. However, the consensus seems to be that restorations built in direct contact to the cavity walls, are in many ways superior to those which are cemented. From the standpoint of durability gold foil is pre-eminent with amalgam a close second. Both of these materials are, neverthless, distinctly limited in their usefulness.

The greatest weakness of preformed restorations is the fact that they must be cemented. There are two disadvantages. First, all of the cementing media, so far produced, are soluble in the oral fluids. Second, these media are friable and weak and tend to fracture or crush under stress. As a result of these properties a hermatic seal is not always maintained, the enamel rods of the cavity margins are not so well supported as in restorations built in direct contact with cavo surfaces.

In spite of its serious shortcomings, the cast cemented restoration has proved to be of great value in restorative dentistry. Time has established that such restorations are fairly durable. The possibilities of anatomically restoring lost tooth substance by the cast restoration is limited only by the ability of the operator. Since all desired anatomical detail can be accurately reproduced in occlusal rehabilitation, it is superior to any other method.

The science and art involved in inlay technique, has been developed to a very high degree. Using present methods, cast restorations can be made with almost unbelievable accuracy. Using as a standard metal objects formed by other means, this may be classified as a precise process.

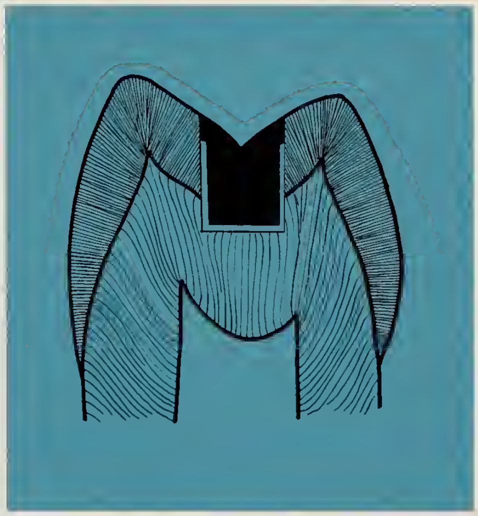
Five steps are involved in the making of a cast restoration, exclusive of cementation and finishing. They are: 1. preparing the cavity; 2. making the wax pattern; 3. making the mold; 4. processing the mold for the reception of the molten metal; 5. making the casting.

Cavity preparation and the making of the wax pattern require great skill and care, and here the art and craftmanship of the operator can be expressed to the fullest extent. Making and processing the mold in the more modern techniques is reduced to the point of utmost simplicity. It is a purely mechanical procedure which can be readily mastered. Making the casting is a procedure which calls for the exercise of considerable skill and judgment in the fusion of the metal. Otherwise, it is also a simple mechanical procedure.

Cementation and finishing are very vital and on these two steps the success of a restoration is largely dependent. If improperly done, a well made and accurate casting may result in a restoration of very questionable value.

The making of a good cast restoration is an involved and difficult process. If a successful result is to be secured, every step must be carried out with painstaking care and accuracy. From the standpoint of durability, the cast restoration is fairly adequate. As a means of anatomically restoring lost tooth structure, it is superior to any other method. In modern dental practice, it is perhaps the most useful restoration we now have.

**ig. 3**
)rawing of a section of a well fitted cast
estoration, which has been relieved to pro-
ide space for cement.



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M.S.D., D.D.S

## GOLD INLAY AND GOLD FOIL

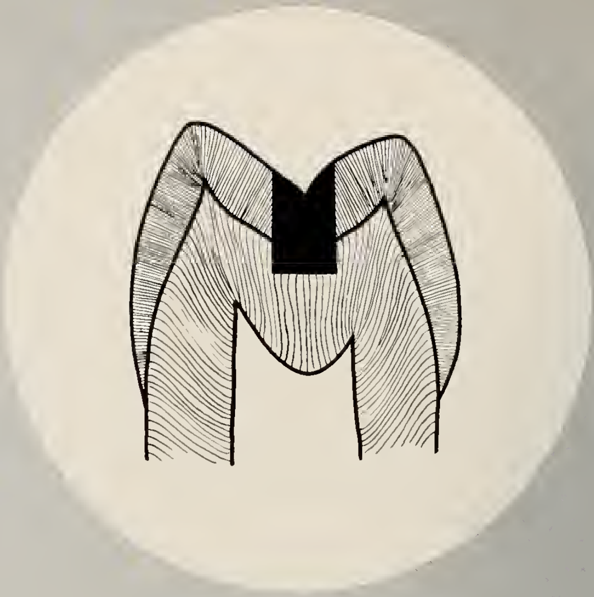


**Fig. 1**

Drawing of a section of a posterior tooth with a metallic restoration (gold foil or amalgam) built in direct contact with the cavity walls. With this type of restoration, the enamel rods at the cavo surface angle are well protected and supported.

**Fig. 4**

Drawing of a cast restoration after cementation. Even when space has been provided for cement, an inlay cannot be completely seated, as shown at (A). (B) shows the margin trimmed flush with the tooth. If this is skillfully done, the cement line can be greatly minimized, however, a slight cement line is always present.

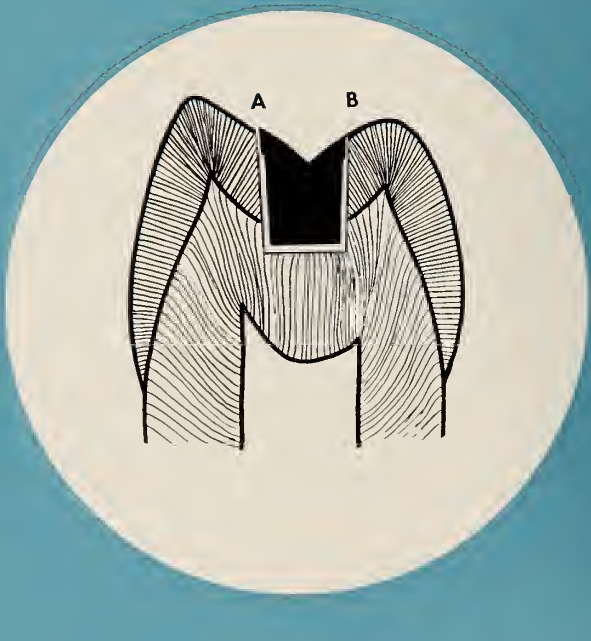


**Fig. 2**

Drawing of a section of a posterior tooth with a well fitted, but uncemented cast restoration in position. Well fitted cast restorations cannot be seated during cementation unless space is provided for the cement.

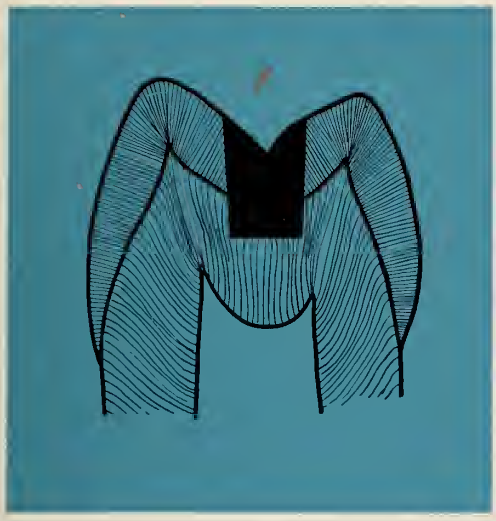
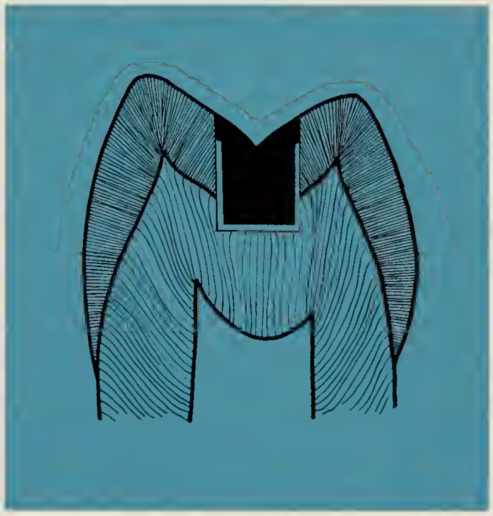


**Fig. 3**

Drawing of a section of a well fitted cast restoration, which has been relieved to provide space for cement.



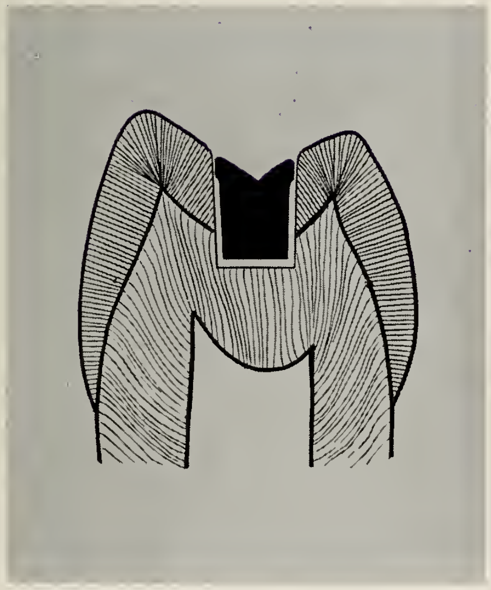


**Fig. 5**

Drawing of a cast restoration which has undergone considerable wear. Beginning failure is now apparent. Cement has superficially disintegrated, leaving margins of cavity and restoration unprotected. Enamel rods have started to break away and margins of restoration are becoming rounded by abrasion. As time goes on, conditions will steadily grow worse, until serious failure results. The greatest need in restorative dentistry today is for better cements. When better cements are produced, cast restorations may closely rival gold foil in durability.

# NOTES

# QUESTIONS

## NOTES

### THE BIOLOGIC ASPECTS OF DENTAL MATERIAL

1. How may the formation of secondary dentin be induced?
2. What is the sequence of changes in the tooth following a filling of silicate cement?
3. What is the effect of an amalgam filling on the dentin and pulp?
4. What are the biologic reactions of the pulp to acrylic resins which are cured in the tooth?
5. What are the advantages of directing a stream of water on the tooth surface during cutting?
6. What are the dangers of allergic reactions to amalgam?

### THE BIO-PHYSICAL PRINCIPLES OF CAVITY PREPARATION

7. Why is an occlusal lock superior to any other type of retention? (Fig. 2)
8. Why is a lingual lock contraindicated in a mandibular anterior tooth?
9. Is a gingival lock indicated in a proximo-occlusal inlay preparation? (Fig. 3) Why? In amalgam? Why?
10. Of two occlusal locks of the same cross-sectional area one is broad and shallow and the other narrow and deep. Which is the stronger?
11. Why should the axio-pulpal line angle be rounded?
12. What is the effect of a rounded pulpal floor on stress distribution in an occlusal preparation?

### SYNTHETIC RESIN FILLINGS

13. How do the self curing resins differ from resins cured by the application of heat?
14. Do all resins shrink during polymerization?
15. Is the resin liquid a toxic material?
16. How do the physical and mechanical properties of resins compare with other restorative materials?

**NOTES**

## QUESTIONS

17. Compare the resistance to wear from attrition of resin restorations with that of other restorative materials.

18. Are future developments likely to produce an improved resin filling material?

### SILICATE CEMENT AND AMALGAM FILLINGS

19. What is the best protective coating to apply to silicate cements after removing the matrix strip?

20. What causes the stained margin in silicate cement restorations?

21. How should a silicate cement restoration be trimmed and polished?

22. Will silicate cements be replaced by the direct resinous filling materials in a somewhat similar manner as acrylic resins replaced hard rubber as a denture base material?

23. What causes an amalgam to discolor tooth tissues?

24. How can this discoloration be prevented?

25. What effect occurs when amalgam and gold alloy restorations are placed in contact?

### GOLD INLAY AND GOLD FOIL RESTORATIONS

26. From a histologic standpoint is the cemented restoration superior to all others?

27. What is the importance of recognizing the linear casting shrinkage of gold and gold alloys used in making cast restorations?

28. In modern procedures the shrinkage effect has been practically eliminated. Does this completely solve the problem of making accurate cast restorations?

29. Is it possible to cement well fitted restorations, disregarding the space which must necessarily be occupied by cement?

30. Is a cement line unavoidable in inlay margins?

31. What hardness of gold alloy is preferable for cast restorations?

# NOTES

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# PREVENTIVE DENTISTRY

**Monday, February 12, 1951**

## OUTLINE

1. Preventive Dentistry:
   Challenges, Objectives, Responsibilities.......A. G. Brodie
   Moderator
2. Public Health Aspects of Preventive Dentistry.......P. E. Blackerby
3. The Role of the Children's Operator in Preventive Dentistry.......K. A. Easlick
4. Caries Control and Prevention: Fact and Fancy.......R. G. Kesel
5. Periodontal Disease in Children.......M. Massler

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# SUMMARY

**PREVENTIVE DENTISTRY: CHALLENGES, OBJECTIVES, RESPONSIBILITIES.** As the profession of dentistry enters the second half of the twentieth century, it finds itself facing new and challenging problems. Until recently, its relationships have been on a person to person basis, i. e., dentist and individual patient. Its training programs, its literature and its professional organizations have been oriented toward this relationship. More than this but largely because of it, dentistry has come to be held, even by its practitioners, primarily as reparative in nature. Practically all dental operations today are directed toward the correction of conditions that have already gone wrong. Such conditions require the direct labor of the hands and the time required for such labor. Thus, the benefits of dentistry are at present limited strictly to the hours available for work on individual patients. Even though the number of dental schools were doubled and each school increased its number of students, there is no possible way of meeting the dental needs of the country on the present basis. The ratio of dentists to population would still be far too low to permit complete coverage. All surveys have led to this conclusion. There is only one alternative—prevention.

For the purpose of our panel, we shall have to use the term Preventive Dentistry rather loosely. Only those diseases for which a specific cause has been proved can be termed preventable. We shall be obliged to think in terms of measures that may minimize the development of deleterious conditions or may be viewed as insurance against such developments. If such measures are found to be effective and do not entail the same amount of time spent on the individual, dentistry will then be able to serve a larger segment of society.

A number of conditions must be met if preventive dentistry is to fulfill the promise it holds.

The first and foremost need is for vigorous prosecution of basic scientific research. The word basic must be stressed because, until the natural processes of the body and of the oral cavity specifically are more clearly understood, we shall lose time on empirical methods of treatment. Like every field of the healing arts, we need above all other things—facts.

Our next need is trained personnel. This means improvement in our educational methods of training dental students, which in turn is dependent upon better trained teachers. Both of these will require further revisions of our dental curriculum with greater emphasis on principles of techniques and basic sciences and less on details. The dental course of tomorrow should strive to become an educational experience directed toward a social service instead of a training course directed toward the servicing of a few broken-down mouths. This will require careful evaluation of every phase of dental education and the establishment of new objectives in place of present biases and traditions.

Finally, we shall need an awakened profession; a profession alive to its social responsibilities and one that keeps abreast of new developments. That the profession is perhaps leading in the effort at change is attested by the widespread interest in the establishment of school, city and state dental clinics devoted to the care of the child. It matters not whether the primary impetus comes from the teacher, the research worker or from the practitioner, so long as it develops. But unless it appears from within the profession it will be forced upon the profession from without, and such an event is not apt to be intelligent.

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PUBLICATIONS: In fields of public health and dental education.

HONORS: Omicron Kappa Upsilon, Delta Omega.

# PUBLIC HEALTH ASPECTS OF PREVENTIVE DENTISTRY

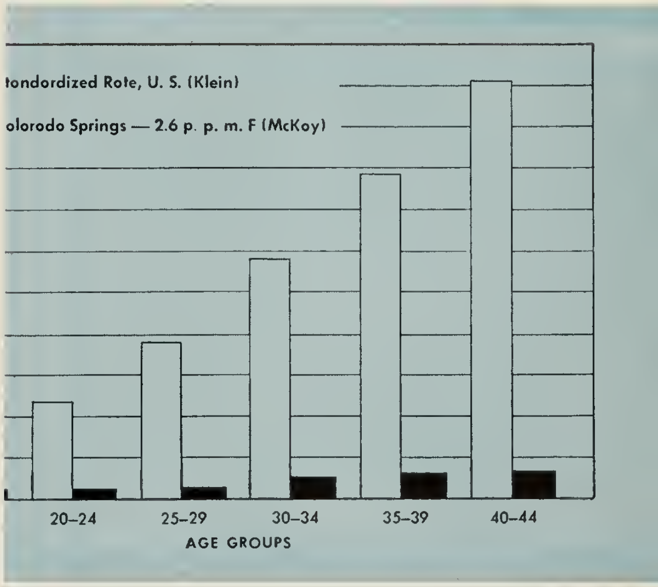
The practicing dentist naturally is interested in that segment of his community's population which represents his own private patients—actual and potential. He is usually less conscious of the dental health problems of the remainder of his fellow citizens. Similarly, the dental practitioner is confronted daily with the reparative and restorative needs of his patients, but it is somewhat more difficult for him to visualize diseases and defects which these same patients may develop in the future and which might be avoided by the timely application of preventive measures.

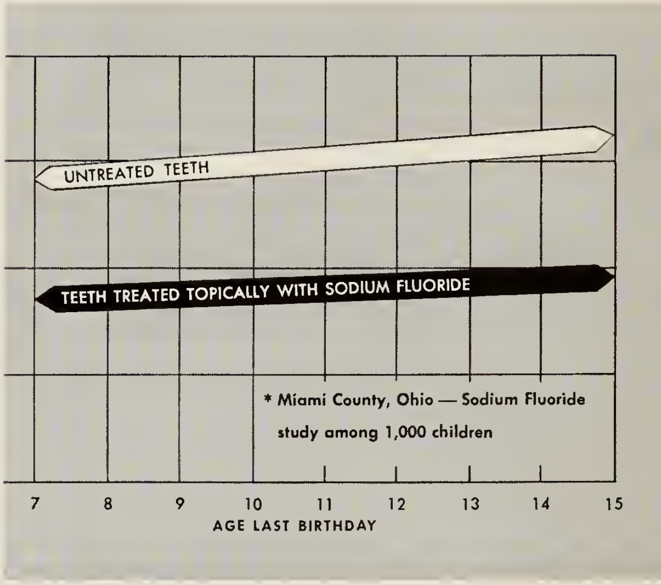
With growing public concern for the health needs of our total population, it becomes more important for every dentist to recognize the public health aspects of his professional responsibility and to develop a sincere interest in the dental health needs of all the people of his community. This is essential not only because he is a dentist, but because he is a citizen who is especially qualified to give leadership in this phase of his community's welfare. Furthermore, as effective measures become available for the prevention of dental disease, it is the practicing dentist who can contribute most effectively to the understanding and utilization of these methods for the benefit of his whole community, as well as of his own patients.

The field of public health is said to encompass those activities that are undertaken for the prevention of disease and the promotion of health which are, primarily, a community responsibility. Thus, the fluoridation of a community water supply represents a public health measure, whereas prophylaxis and the topical application of sodium fluoride by the private practitioner are examples of the individual approach to prevention of dental disease (Fig. 1). Both methods are essential for a good community dental health program, and much more can be accomplished through the joint efforts of public health workers and practicing dentists than can be expected from either group working alone. The dramatic results which have been achieved in the control of smallpox, diphtheria, pneumonia, tuberculosis and other communicable diseases could not have been brought about without the combined efforts of private practitioners and public health agencies, and interested lay groups as well. In dentistry, the same is true: the dental health problems of the whole community, as well as those of our individual patients, must have the interest and attention of every dentist, and we must join hands with others in our communities for the promotion of health and the prevention of disease (Fig. 2).

Finally, our accomplishments in the prevention or control of dental disease can be measured and demonstrated more satisfactorily on a community basis than in the private dental office. Morbidity and mortality rates have useful application in dentistry, too, but they must be applied to large population groups to have reliability and statistical significance. Thus, the establishment of dental caries prevalence rates and tooth mortality rates, for example, can provide valuable criteria of dentistry's progress, not only in research but in the application of knowledge for the day-by-day improvement of community health (Figs. 3 and 4).

oth Loss Rate at Colorado Springs, Colo-
ardized Rate." (Courtesy—Dr. H. T. Dean,
of Dental Research).

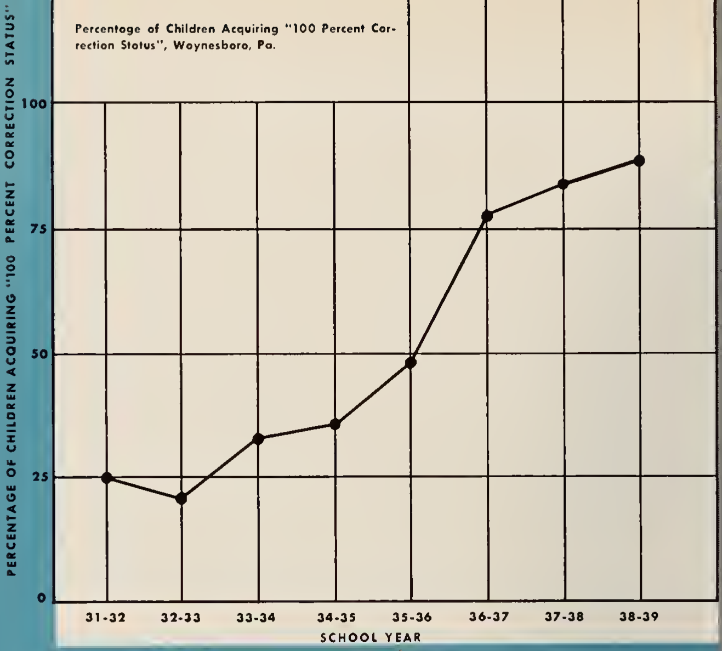




Fluoride Program
ty. (Courtesy—Dr.
I. S. Public Health

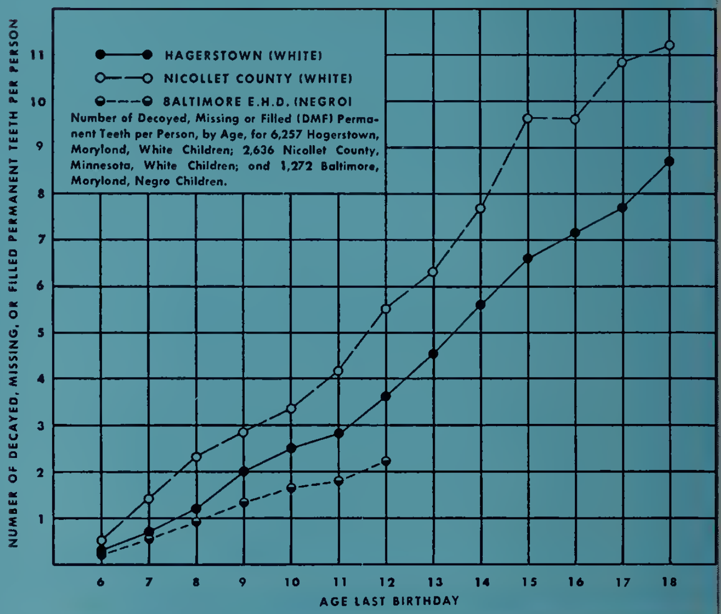
PHILIP E. BLACKERBY, JR.
A.B., D.D.S., M.S.P.H

# PUBLIC HEALTH ASPECTS OF PR



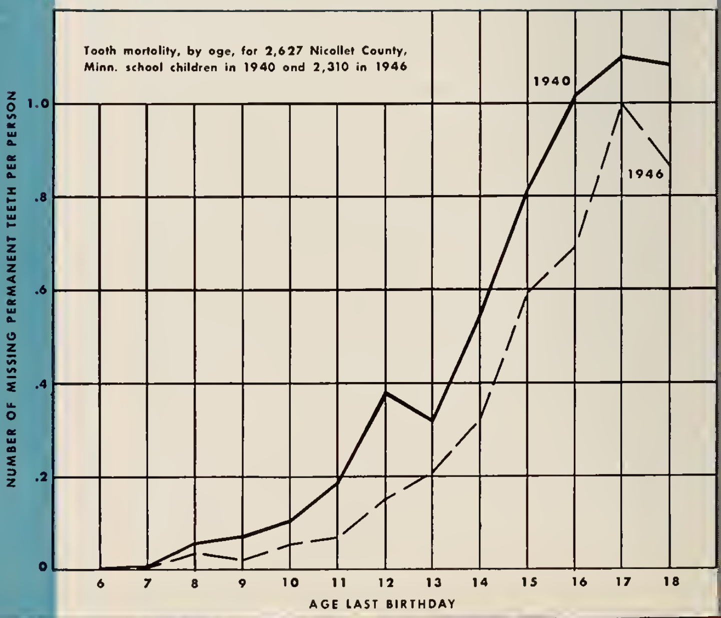
Percentage of Children Acquiring "100 Percent Correction Status", Waynesboro, Pa.

**Fig. 2**



Number of Decayed, Missing or Filled (DMF) Permanent Teeth per Person, by Age, for 6,257 Hagerstown, Maryland, White Children; 2,636 Nicollet County, Minnesota, White Children; and 1,272 Baltimore, Maryland, Negro Children.

**Fig. 3**



Tooth mortality, by age, for 2,627 Nicollet County, Minn. school children in 1940 and 2,310 in 1946

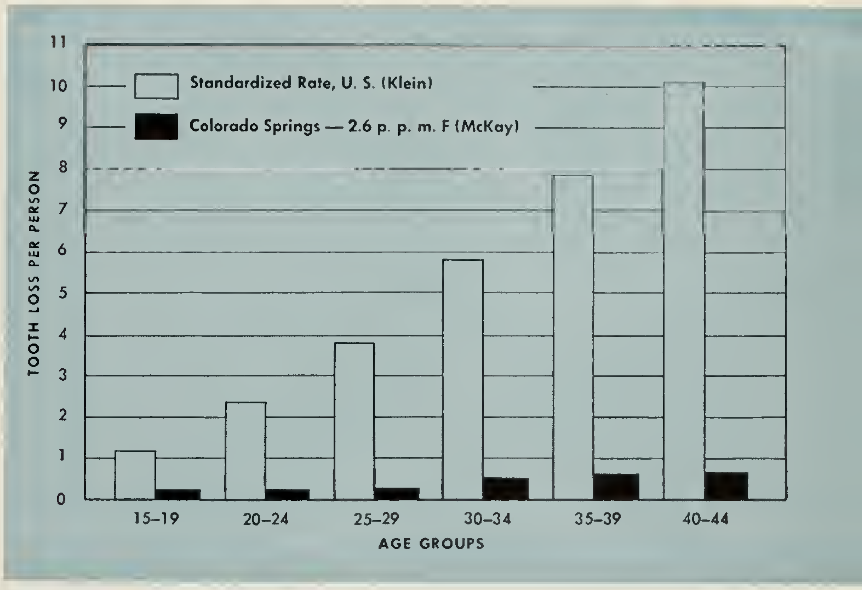
**Fig. 4**

**Fig. 1a**

Water intake for Colorado Springs, Colorado, Ruxton Plant.

**Fig. 1b**

Comparison of Tooth Loss Rate at Colorado Springs, Colorado, with "Standardized Rate." (Courtesy—Dr. H. T. Dean, National Institute of Dental Research).



## PUBLIC HEALTH DENTISTRY

## PRIVATE DENTISTRY

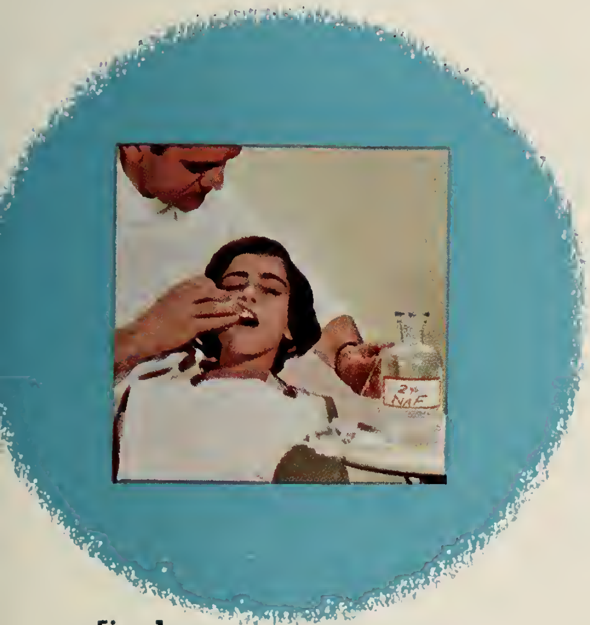


**Fig. 1c**

Topical application of 2% Sodium Fluoride by the dentist.

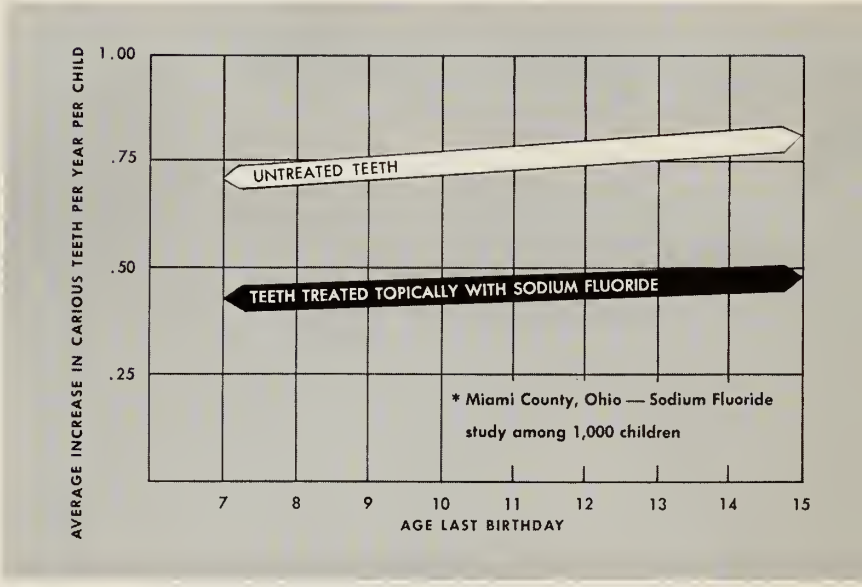


**Fig. 1d**

Results of Sodium Fluoride Program in One Community. (Courtesy—Dr. J. W. Knutson, U. S. Public Health Service).

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PREVENTIVE

DENTISTRY

Charts (left) courtesy of W. J. Pelton and J. M. Wisan, *Dentistry in Public Health.*

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HONORS: Phi Kappa Phi, Omicron Kappa Upsilon.

# THE ROLE OF THE CHILDREN'S OPERATOR IN PREVENTIVE DENTISTRY

The practice of operative dentistry for children may be designated as preventive dentistry at least in a limited sense. Intriguing technical vistas unfold for the alert children's operator if the following sequence of preventive measures by timely and scientific interventions are granted:

1. An incipient cavity from becoming a deep cavity in a broken down crown.
2. A deep cavity from becoming an exposed vital pulp.
3. An exposed vital pulp from becoming a putrescent root canal.
4. A putrescent root canal from becoming a periapical region of infected bone.
5. A periapical area of infected bone from becoming a cyst.
6. The combination broken down crown, putrescent root canal and periapical cyst from becoming a space.
7. The space from creating tissue injury, malocclusion, or an emotional hazard for a young person.

Sufficient preventive technics are now available to the operative dentist. He can prevent in individual patients incipient enamel decalcifications from becoming cavities in the dentin if he utilizes sufficient motivation in his chair-side teaching. When such persuasive motivation cannot be developed, he can eliminate caries involvement of dental pulps and a long chain of sequellae by a careful study of annual bite-wing radiographs. Beginning proximal calcification can then be detected and can be promptly and thoroughly filled.

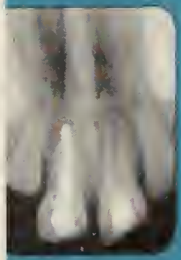
The freshly cut dentin in cavity preparations requires special consideration. Unscientific cutting and medications leave young teeth most uncomfortable. Discriminatory use of revolving instruments and the careful selection of drugs, varnishes and bases make filled teeth more comfortable.

Problems of diagnosis arise as intervention in the caries process is delayed. Symptoms are most difficult to assess or estimate accurately when the children's operator is seeking a decision whether to attempt pulp capping or partial pulpectomy for a young tooth. It is quite impossible for the operator to determine the actual extent of degenerated pulp tissue accompanying a carious exposure of the pulp chamber. However, the amputation and removal of the entire coronal pulp and the protection of the root stumps with calcium hydroxide provides much more assurance that healthy tissue is being capped than when calcium hydroxide is placed over an exposed area of the pulp in a deep cavity.

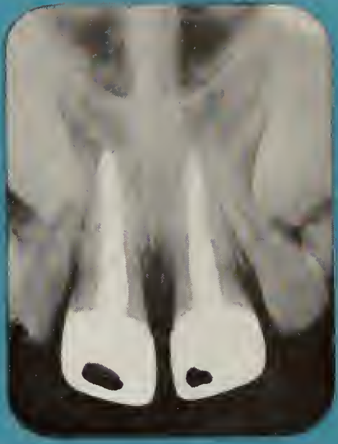
Scientifically satisfactory technics are available for filling root canals, provided two successive bacteriologically negative cultures have been obtained. For example, there are available silver root canal points to fill the root canals of nonvital young permanent molars and "tailor made" sections of gutta percha to fill the root canals of young permanent incisors.

There are no technics available for treating and filling nonvital primary molars which will satisfy the requirements of a scientific endodontist. Health considerations demand extraction, presenting a problem of space management.

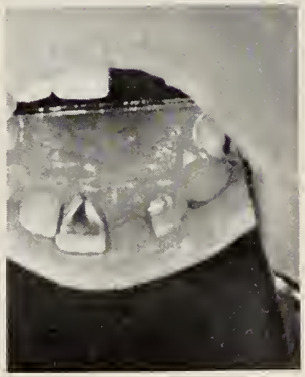
Experimentation has supplied the children's dentist with technics which can preserve almost every injured incisor. Periapical curettage, to remove an area of granulation tissue or a cyst, provides a technic with which to preserve many injured, but neglected young incisors. No longer is it necessary to extract incisors with fractured roots. Unless the fracture line communicates with the gingival sulcus, stabilization of the tooth usually permits a callus to form and bony healing to take place in a remarkably short time. Good results can be obtained even with the young permanent incisor completely removed by trauma. If replanted promptly, you may reasonably expect to avoid a hazard to esthetics, health, occlusion and, perhaps, a child's emotional well-being.



trescent incisors
(right) for 2½



(a) the radiographs



bridge assembled
(mirror photograph)

**KENNETH A. EASLICK**

**A.M., D.D.S**

# THE ROLE OF THE CHILDREN'S

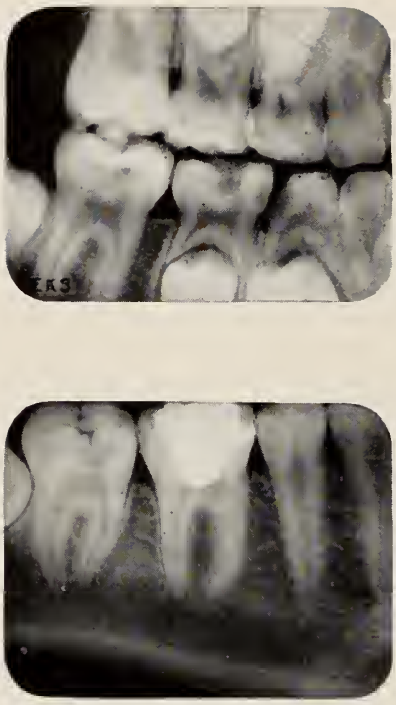


**Fig. 1**
A 5-year check of a partially pulpectomized 1st permanent molar. The calcium hydroxide has stimulated the deposition of a dentin plug in both roots.

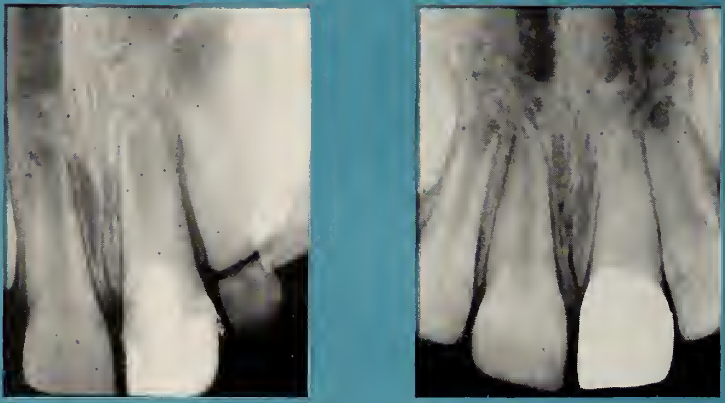


**Fig. 2**
A 2-year check of a fractured incisor treated by performing a partial pulpectomy and capped with calcium hydroxide.

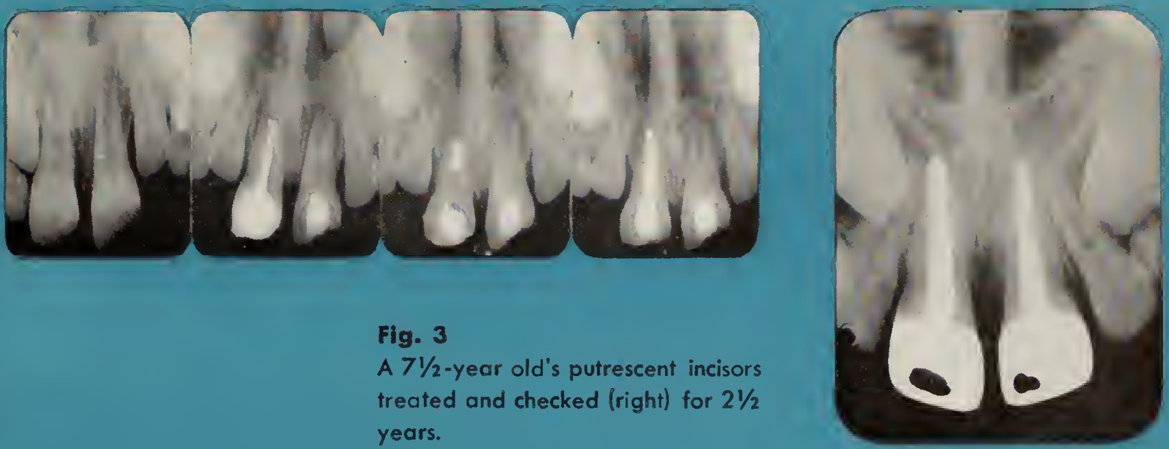
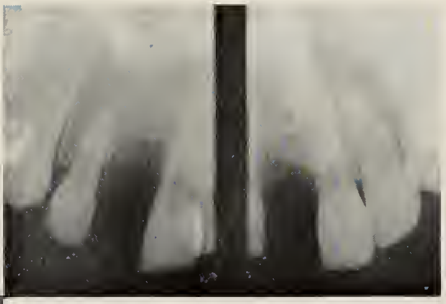
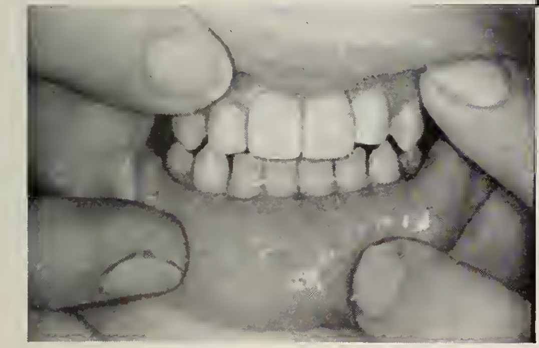


**Fig. 3**
A 7½-year old's putrescent incisors treated and checked (right) for 2½ years.

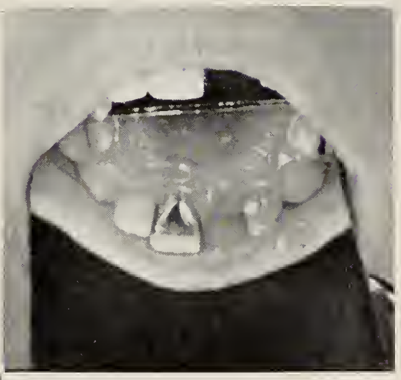
**Fig. 4**
Replantation of the central being too late for a successful outcome, a bridge is constructed at 11 years 7 months:



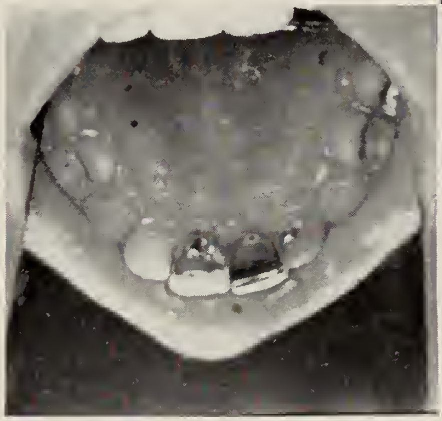
(a) the radiographs



(b) space restored



(c) castings (mirror photograph)



(d) bridge assembled (mirror photograph)

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## CARIES CONTROL AND PREVENTION: FACT AND FANCY

Many methods have been suggested for controlling dental caries but few have proved effective. The multiplicity of ideas has resulted from the indefinite understanding of caries etiology. Recent years have produced a clearer concept of the destructive forces that destroy tooth tissue. An accumulation of chemical, bacteriological and dietary evidence has emphasized the importance of acid production (in contact with the tooth surface) by the bacterial action on readily fermentable carbohydrates.

As yet, however, no easy way has been developed for caries prevention and control. The accepted methods that have been established as effective in clinical studies are either time consuming or unpalatable. They either require the individual to deny himself something (sugar) that he greatly enjoys or ask him to perform consistently and conscientiously a time consuming operation (tooth brushing or topical applications.) The search continues for something that can be introduced into the routine living conditions that will not require thought, action or denial.

The fluoridation of drinking water may be an excellent partial solution to this quest. While sufficient controlled evidence is not yet available to prove that artificial fluoridation is as effective in reducing caries activity as is natural fluoridation, (40-50 per cent average community reduction) presumptive and preliminary evidence indicates that it will.

There are a few methods that controlled clinical studies have proved will reduce the incidence of dental caries. The most effective is the moderation or restriction of sugar consumption at meal time and the elimination of eating sugar-containing substances between meals. This way to caries control is difficult because of the popularity of sugar.

## CARIES CONTROL

| | | ADVANTAGES | DISADVANTAGES |
|---|---|---|---|
| **ACCEPTED** | 1. Reduce sugar intake (especially between meals) | a. Caries reduction<br>b. Improved nutrition | a. Mass cooperation difficult<br>b. Economy |
| | 2. Brush teeth and/or rinse mouth immediately after eating | a. Caries reduction<br>b. Clean mouth | a. Inconvenience<br>b. Rigid discipline required |
| | 3. Topical application of 2% sodium fluoride solution | a. Caries reduction<br>b. Simplicity | a. Not a mass control method<br>b. Requires dental appointments |
| **PROMISING** | 1. Fluoridation of water | a. Caries reduction (?)<br>b. Mass control method | Not yet proved |
| | 2. Ammoniated dentifrice | a. Caries reduction (?)<br>b. Use of "natural" antibiotic | Rigid discipline required |
| | 3. Tooth impregnation<br>a. Zinc chloride<br>b. Silver nitrate | a. Caries reduction (?)<br>b. Patient cooperation unnecessary | a. Causticity<br>b. Discoloration<br>c. Time consuming |
| | 4. Penicillin dentifrice | Caries reduction (?) | a. Sensitization<br>b. Drug fastness<br>c. Discipline required |
| | 5. Chlorophyll dentifrice | Caries reduction (?) | Rigid discipline required |

| | | | |
|---|---|---|---|
| **UNACCEPTED** | 1. Calcium therapy | **BECAUSE** | 1. Caries not related to calcium metabolism |
| | 2. Vitamin therapy | | 2. Caries is a dietary, not nutritional disease |
| | 3. Fluoride tablets | | 3. Not absorbed by completed enamel |
| | 4. Synthetic Vitamin K | | 4. Toxicity |
| | 5. Glyceric aldehyde | | 5. Toxicity, interferes with metabolism |

Tooth brushing diligently done soon after eating will reduce caries activity. Again the need for the conscientious development of an inconvenient habit reduces the practical effectiveness of the method.

The topical application of a two per cent sodium fluoride solution to the clean dried tooth surface is another established preventive procedure. Sufficient evidence has been reported by various investigators to justify the acceptance of this means for caries control.

There are several other promising methods. They, however, have either not been tested over a long enough time in controlled studies on sufficient numbers of people to establish their validity, or the possibility of their hazardous effects has not been eliminated.

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HONORS: Sigma Xi, Omicron Kappa Upsilon, Member Italian Medical Nutrition Mission, 1945.

# PERIODONTAL DISEASE IN CHILDREN

Preventive dentistry has given little attention to the problem of periodontal disease. Yet periodontal disease ranks second only to dental caries in the destruction of the dental apparatus.

While caries is primarily a disease of childhood, it has been *assumed* that periodontal disease does not exist in the child and occurs only in the adult. However, recent epidemiological studies, using a quantitative method of assessment called the P-M-A Index (page 88), have revealed, surprisingly, that periodontal disease *does* exist in children—and to a remarkable degree.

It was found that while 40% of the children between the ages of 5 and 14 years were clinically free of any evidence of even slight gingival inflammations (ideal gingivae), 60% of the children showed some evidence of gingivitis. In approximately 35% of this latter group, the inflammations were very mild and confined to 1 to 4 of the anterior gingival papillae (very mild gingivitis). We may categorize both groups (75% of the total) as having "normal" gingivae.

What was most surprising is the fact that about 17% of the children showed a very severe and clinically abnormal gingivitis. (In 8%, the gingivae were questionable.) This high figure of almost 1 child in 6 showing distinct, clinical evidence of periodontal disease negates the erroneous hypothesis that periodontal disease does not exist in children. It does.

The great majority of simple, mild gingivitis found in children between 5 and 14 years of age is obviously related to eruption and physiologic crowding of the teeth that occur during the mixed dentitional period. These gingivitides are *transitory and self-corrective*. The gingivitis tends to disappear as the teeth complete their eruption and are molded into

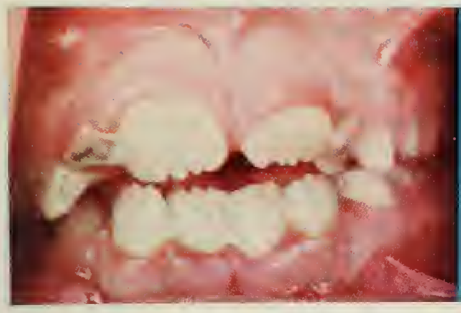


**Fig. 1**

**"Eruption" Gingivitis.** Note the characteristic marginal color.

the final arch form of the young adult. Figures 1 and 2 show the transitory type of eruption and of crowding gingivitis. Eruption gingivitis is generally marginal and hyperemic in character. The crowding gingivitis is almost always papillary, swollen and edematous in character.

On the other hand, 17% of school children show a clinically (and histologically) pathologic condition of the gingiva. These are not transitory and *not* self-reparative. Unless these severe gingivitides are corrected by the dentist, they will probably lead to severe periodontal disease in the adult. Figure 3 shows a type of gingival hyperplasia which usually accompanies severe gingivitis during the teen-age. This is *not* self-corrective. It has been assumed, but not yet proved, that the underlying factor may be a transient endocrine imbalance which occurs during puberty and adolescence.

Figure 4 illustrates a condition termed gingivosis. This consists of a marked destruction of the papillary and marginal gingiva which resembles (but is not) a "Vincent's" infection. Gingivosis is commonly seen (33%) in severely malnourished children (Schour and Massler, Am. J. Orthod., Oct., 1947).

The graph on the following page shows the epidemiological trends in the prevalence of gingivitis from birth to old age. This graph illustrates the fact that gingivitis is a *biphasic* phenomenon. It emphasizes the fact that periodontal disease in children is not identical with the disease of the adult. In general, gingivitis seen in childhood is acute in type and transitory in nature. It is not cumulative but tends to be self-reparative. Periodontal disease in the adult, on the other hand, is cumulative and *not* self-reparative in nature.

Notice that the curve does not fall to zero after adolescence but forms a plateau from 18 to 28 years. This plateau, which includes the severe, non-reparative gingivitides seen in childhood, probably forms the base-line for the chronic cumulative condition during adulthood. Further clinical studies of a longitudinal nature are essential before the problem can be clarified further.

All these preliminary findings lead to the inevitable conclusion that the prevention of periodontal disease merits the same scientific attention and financial support that have been given to the problem of dental decay.

**Fig. 3**

**"Hyperplastic" (Fibretic) Gingivitis.** An adolescent mouthbreather.

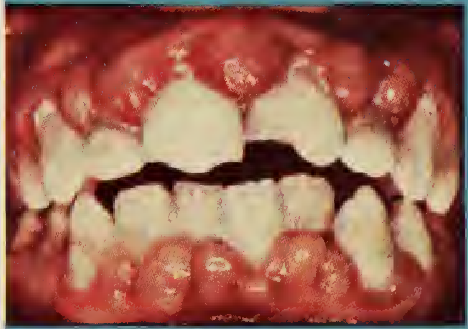
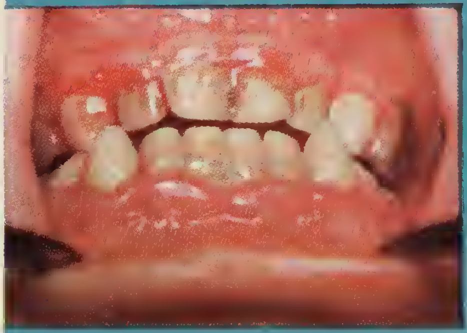


**Fig. 4**

**Gingivosis.** A malnourished child (3-12 years of age).



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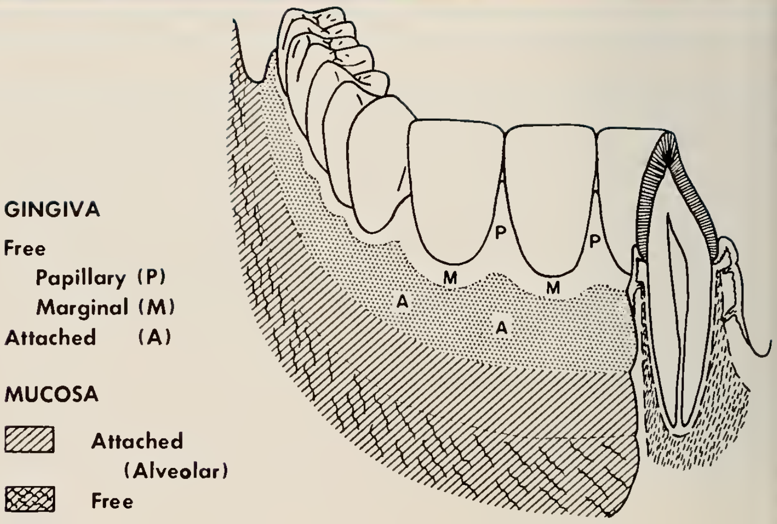
UBLICATIONS: Research in nutrition, dental caries, histology,

## PERIODONTAL DISEASE IN C

## PREVALENCE OF GINGIVITIS

| | | | | |
|---|---|---|---|---|
| **FIRST PHASE**<br>ACUTE<br>TRANSITORY<br>SELF-REPARATIVE | **AGES 6-16 YEARS**<br>PEAK 12-14 YEARS | | | |
| | NORMAL | | ? | ABNORMAL |
| | NONE | MILD | MODERATE | SEVERE |
| | 40% | 35% | 8% | 17% |
| **SECOND PHASE**<br>CHRONIC<br>CUMULATIVE<br>PERMANENT | **AGES 25-50 YEARS**<br>PEAK OVER 50 YEARS | | | |
| | NORMAL | | ? | ABNORMAL |
| | NONE | MILD | MODERATE | SEVERE |
| | 20% | 50% | 13% | 17% |



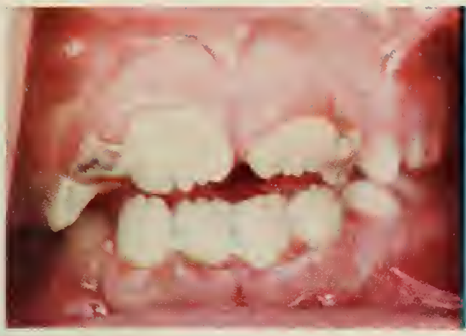


**Fig. 1**

**"Eruption" Gingivitis.** Note the characteristic marginal color.

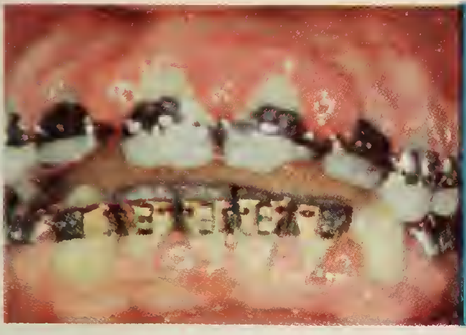


**Fig. 2**

**"Crowding" Gingivitis.** Condition disappeared after orthodontic treatment was applied.

**Fig. 3**

**"Hyperplastic" (Fibretic) Gingivitis.** An adolescent mouthbreather.

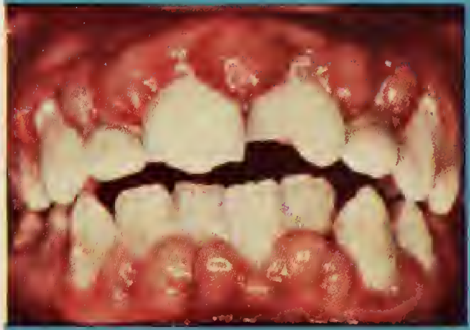


**Fig. 4**

**Gingivosis.** A malnourished child (3-12 years of age).

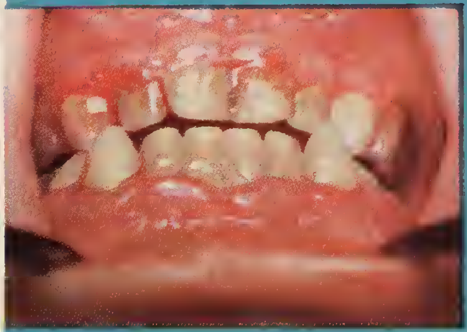
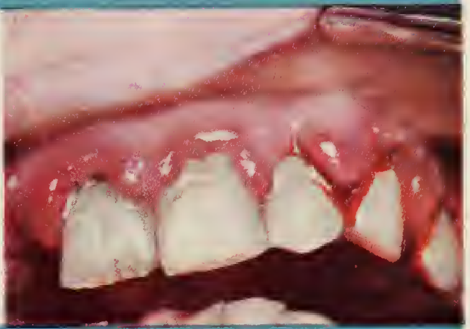


**Fig. 5**

**Gingivosis.** A malnourished man (60 years of age).



d 5

PREVENTIVE

DENTISTRY

# NOTES

# QUESTIONS

## NOTES

### PUBLIC HEALTH ASPECTS

1. What is meant by DMF and DMF rates? Tooth mortality rates?
2. Why should the practicing dentist be interested in the establishment of a dental public health program in his community?
3. Is public health dentistry concerned only with the dentally indigent? Only with children?
4. What resources are available to assist dentists or dental societies in the development of local dental public health programs?
5. What percentage of the total population is now believed to be receiving adequate dental care? The child population?
6. How may preventive dental services be made available to more of our people?

### THE ROLE OF THE CHILDREN'S OPERATOR

7. How frequently should posterior bite-wings be secured for the child patient?
8. What cavity medication makes for comfortable teeth after the fillings have been placed?
9. Why is calcium hydroxide selected for pulp capping or vital partial pulpectomy technic?
10. Why cannot infected primary molar teeth be treated and filled scientifically?
11. How is retention obtained for castings which restore the fractured incisal edge of teeth?
12. How is a tooth "replanted"?

NOTES

## QUESTIONS

### CARIES CONTROL

13. How effective is topical fluoride treatment when applied to the teeth of adults?

14. Can tooth brushing be effective in controlling dental caries?

15. What are some of the mass caries control methods?

16. What are some of the factors which initiate enamel caries?

17. What are some of the factors involved in the rate of progress of dental caries?

### PERIODONTAL DISEASE IN CHILDREN

18. Are we ready for a program of prevention in periodontal disease?

19. Differentiate between periodontal disease and gingivitis—in the child and in the adult.

20. List the clinical characteristics of gingivitis in (a) the grade-school child, (b) the teen-age child and (c) the adult.

21. When is gingivitis "normal"?

22. Explain why gingivitis is absent from birth to five years of age.

23. What is "rampant" gingivitis?

24. What is the relation of caries and gingivitis in the child?

25. Is it true that in the adult, caries and periodontal disease are antagonistic?

26. What is gingivosis? What are its sequelae? Under what circumstances does it occur?

# NOTES

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# DISEASES OF THE MOUTH

**Monday, March 12, 1951**

## OUTLINE

| | |
|---|---|
| 1. Oral Diagnosis | B. G. Sarnat |
| **Lesions Local in Origin** | Moderator |
| 2. Dental Aspects | H. B. G. Robinson |
| 3. Bacteriologic Aspects | J. L. T. Appleton |
| 4. Otorhinological Aspects | F. L. Lederer |
| **Lesions Systemic in Origin** | |
| 5. Dermatologic Aspects | F. E. Senear |
| 6. Hematologic Aspects | C. V. Moore |

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## SUMMARY

**ORAL DIAGNOSIS.** Many patients will come initially to the dentist to seek advice when they become aware of lesions of the lip, buccal mucosa, tongue, floor of the mouth, hard palate and gingiva. In addition to these patients, there are many more who will visit the dentist for routine periodic oral examination and treatment. Among these may be individuals who show early stages of lesions of which they are unsuspecting.

If oral diagnosis is to be accurate, the technique of obtaining the patient's history and the oral examination must be careful and complete. The systematic examination of the oral cavity as part of physical diagnosis has been a neglected phase in dental practice. Too frequently the oral examination by the dentist consists only of an inspection of the physical aspect of the teeth, their mutual relation and the occlusion of the dental arches. It should not pertain to the teeth alone but to all of the oral tissues and associated structures. Particular areas such as the submaxillary, submental and lower cervical regions should also be subjected to careful examination. In addition, certain special examinations are frequently necessary to arrive at a final diagnosis.

Obtaining the patient's history is an essential step in oral diagnosis. It is an art and science which merits meticulous attention in every case. Relevant and significant facts are often either overlooked, forgotten, or withheld by the patient unless they are drawn out by careful and systematic questioning. It behooves the dentist who first sees the patient with the complaint of a sore in the mouth to listen attentively, patiently and understandingly and to take sufficient time to question him systematically and thoroughly.

Obtaining a written record of the patient's history has become axiomatic in the practice of medicine. It is equally important in oral diagnosis. This is particularly true as advances are made on the significance of changes in the oral mucosa as indicators of the health and disease of the individual.

Oral examination and special examinations are described and illustrated in the supplementary chart on these subjects.

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## DENTAL ASPECTS

Local factors are often overlooked in the search for causes of lesions of the oral mucosa. The oral mucosa is almost constantly under bombardment by physical, chemical and thermal irritants. Inflammatory reactions result which may be productive or destructive in nature. The lesion will vary in accordance with the nature of the irritant, the length of time it is applied, its severity and the reaction of the individual tissues.

Among the many causes of trauma in the oral tissues are the following:

a. sharp edges of natural teeth or of restorations,
b. the combination of negative pressure in the oral cavity and occluding surfaces of teeth,
c. tooth picks, toothbrushes and other agents designed for oral hygiene,
d. coarse food and sharp bones,
e. objects placed in the mouth through habit, as boby-pins, needles and tacks.

Any of these traumatic conditions may result in hyperemia, edema, ulceration, granulation tissue, hypertrophy and hyperplasia, hyperkeratosis and other reactions.

From the chemical standpoint there may be direct irritation with solution of cell membranes or of their protective coverings, modifications of osmotic relationships, alteration of electrical potentials, or precipitation of proteins. These reactions may be produced as direct "burns" or as alterations in the "sensitivity" of tissues. Irritating drugs such as aspirin, phenol, silver nitrate or chromic acid, certain foods, condiments, tobacco products, alcohol, restorative materials, dentifrices, mouth washes, bacterial toxins and other agents may all produce injury to

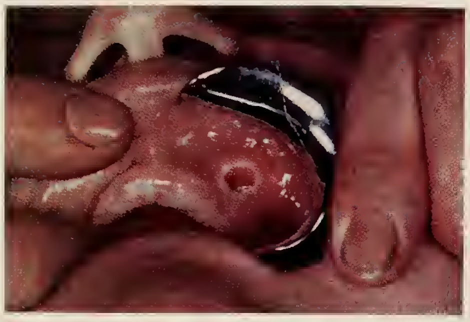
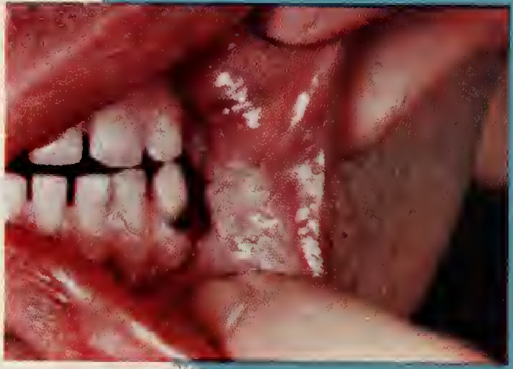


**Fig. 2**

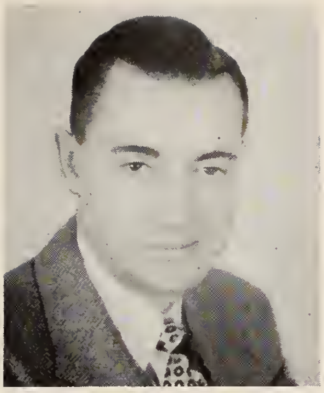
Cheek chewing lesion with coarse fibrinated appearance of surface epithelium. In this particular instance the cheek chewing was a result of a habit. In many cases, the combination of heavy cheeks and negative pressure during inspiration may lead to similar lesions occurring bilaterally.



the oral mucosa. Many different types of lesions may be encountered varying from the erythema of contact (allergic) stomatitis under denture materials to the necrotic regions when tissues are destroyed by acids.

A number of miscellaneous conditions must be considered in any discussion of local oral conditions. Disturbances in growth including Fordyce spots (ectopic sebaceous glands), median rhomboid glossitis, fissured tongue, torus palatinus and torus mandibularis are sometimes misdiagnosed. Diseases of unknown cause or possibly of psychosomatic origin include burning tongue, benign migratory glossitis (geographic tongue), and lichen planus. These may be mistaken for signs of pernicious anemia, vitamin deficiency, neoplastic growth or trauma.

The alert dentist is aware of the possibility of occurrence of the various stomatites and glossites of local origin. He is also aware of those of systemic origin and considers them in the examination and diagnosis of oral lesions. He weighs the possibility of local causes preparing the tissue for invasion by bacteria, virus or other organic forms and against the lowering of local resistance by general systemic factors. In designing his therapy he seeks the underlying cause and does not invite failure or recurrence by treating secondary infection while allowing trauma to remain. The well trained dentist can differentiate lichen planus, leukoplakia and cheek chewing. He knows when he needs the biopsy to aid him in completing his diagnosis and preventing incorrect treatment. The modern, well trained and intelligent dentist has assumed his full role as the diagnostician of all oral disease.



**HAMILTON B. G. ROBINSON,**
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R: Co-Editor, Year Book of Dentistry, 19

NS: Oral Pathology. Editor, Journal of De
ciate Editor, Oral Surgery, Oral Medicine a

## DENTAL ASPECTS

**Fig. 3**

Aspirin burn. Aspirin (acetylsalicylic acid) tablets held against the mucosa may produce discrete lesions, powdered tablets may produce diffuse lesions. The acid actually burns the mucosa and the lesions are treated as any intraoral burn.

**Fig. 4**

Benign migratory "glossitis" (geographic tongue, wandering rash). The papillae of the tongue have been lost in more or less semilunar or ovid areas along the lateral borders of the tongue. These regions are outlined by gray or white margins, and the central areas are increased in redness. The lesions are usually painless but an accompanying burning tongue may result from psychoneurotic changes, especially cancer phobia.

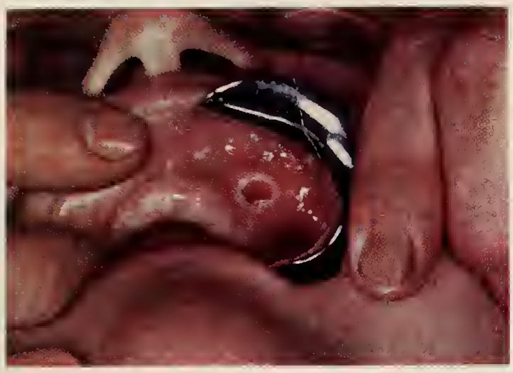
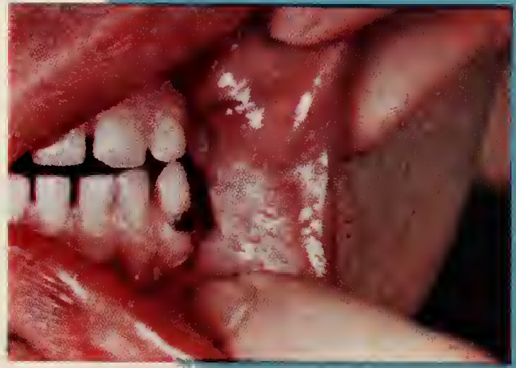


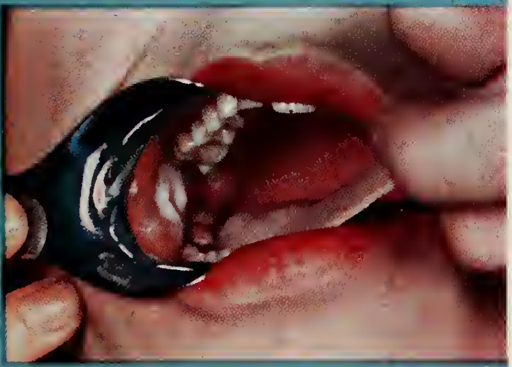
**Fig. 1**

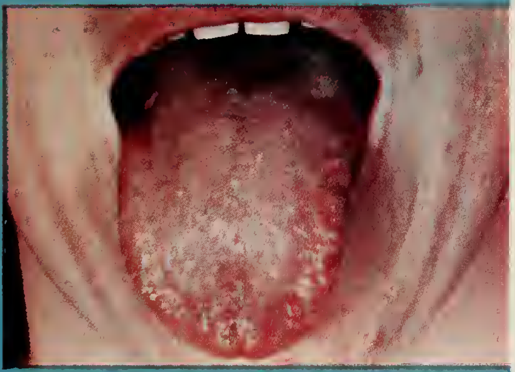
Denture sore (granuloma fissuratum, decubital ulcer) resulting from physical trauma produced by an impinging denture border. Pressures from improperly constructed dentures or from dentures that have outlived their usefulness may result in either hypertrophy and hyperplasia or in ulceration and necrosis. Granulation tissue at the edge of the ulcer may simulate a neoplasm.

**Fig. 2**

Cheek chewing lesion with coarse fibrinated appearance of surface epithelium. In this particular instance the cheek chewing was a result of a habit. In many cases, the combination of heavy cheeks and negative pressure during inspiration may lead to similar lesions occurring bilaterally.







**JOSEPH L. T. APPLETON,**
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AUTHOR: Infection with Special Reference to Dental Practice. Editor of Control of Pain in Dental Practice.

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HONORS: Phi Beta Kappa, Sigma Xi, Omicron Kappa Upsilon, Callahan Medal, Past President International Association for Dental Research, Fellow American Association for the Advancement of Science, Fones Award.

## BACTERIOLOGIC ASPECTS

Before birth the oral cavity is sterile. During or shortly after birth, a variety of bacteria pass into the mouth and come to rest on the new mucosa. Some of these, already adapted to the conditions encountered, survive and multiply. Some of the survivors "gang up" in symbiotic and mutually advantageous relationships. Others succumb in the struggle for existence. There is gradually established a "resident" flora. This is not static or final, but is subject to constant disturbance by the introduction of new strains and species and by the sequence of conditions which occur in the mouth. The "resident" flora is supplemented by a "transient" flora, presumably of minor importance in the economy of the mouth. In fact, however, members of the "resident" flora probably disappear to be replaced by strains or species which previously had been looked upon as transients.

Qualitatively and quantitatively, the resident flora differs in different individuals and in the same individual under different conditions. Representatives of bacteria, yeasts, protozoa, and viruses are frequently found. Among the factors or conditions which influence the kinds and numbers of mouth bacteria are:

1. the condition of the dentition and the gingiva
2. the "clearance mechanism"
3. the diet
4. the saliva
5. antibiosis and symbiosis
6. chemoprophylactic and chemotherapeutic agents

To approach an understanding of the nature and significance of the micro-organisms of the mouth, it is necessary to consider also the micro-

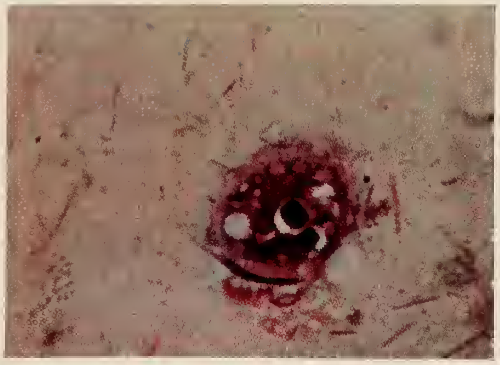


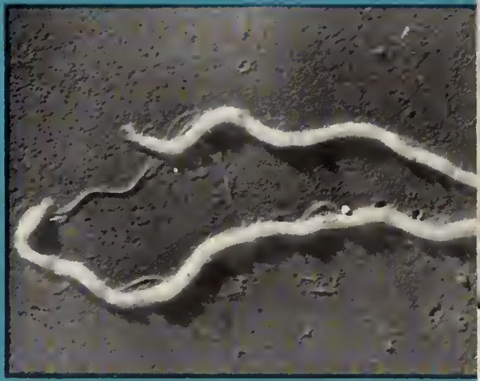
**Fig. 3**

Endamoeba gingivalis in mouth smear, carbol fuchsin and methylene blue. Magnification, Ca. 950X.

organisms of the upper respiratory tract (nasal cavity and pharynx).

The role of intra-oral micro-organisms is not limited to the pathogenesis of intra-oral disease. Even their potential role in focal infection does not exhaust their significance. Three areas —mouth, nasal cavity and pharynx—occupy a strategic position at the entrance to, and at the crossroads of the respiratory and alimentary tracts. This strategic position relates the respective microbial populations to the initiation of respiratory, enteric, and wound infections. This applies not only in the individual host but also in other human beings by way of transmission through the air and of other transmitting mechanisms.

**Fig. 6**



Treponema pallidum 7200X, enlarged to 17,000X. Electron photomicrograph. (Courtesy E. S. Hampp, D.D.S., A.D.A. Research Associate at the National Institute of Dental Research)

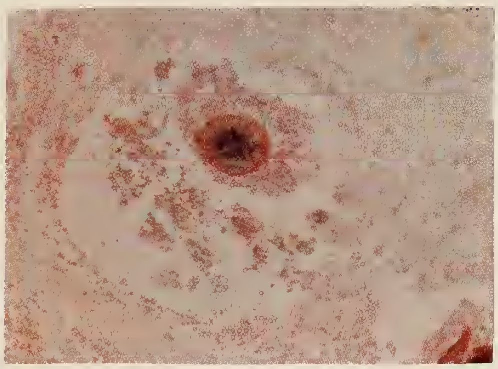


**Fig. 5**

Actinomyces bovis in tissue obtained for biopsy, hematoxylin and eosin. Magnification, ca. 450X.

**JOSEPH L. T. APPLETON, B.S., D.D.S.**

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# BACTERIOLOGIC ASPECTS

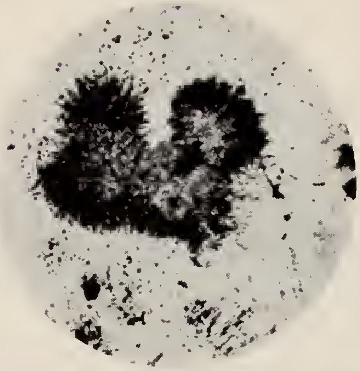

**Fig. 1**

Actinomycosis of pulp of upper second molar. (Abb. 284, S. 223 of Euler U. Meyer, 1927, Pathohistologie d. Zaehne.)

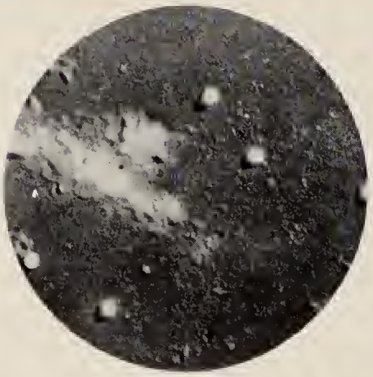

**Fig. 2**

Herpes. Simplex. Vesicle fluid from lip. Gold shadowed, electron micrograph. Magnification 20,000X. (Courtesy of T. McNair Scott, M.D., Children's Hospital, Philadelphia, Pa.)

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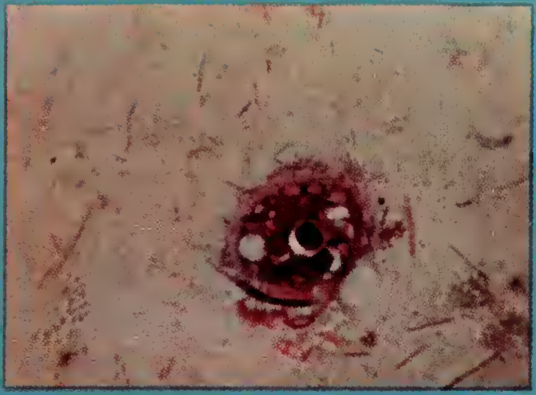

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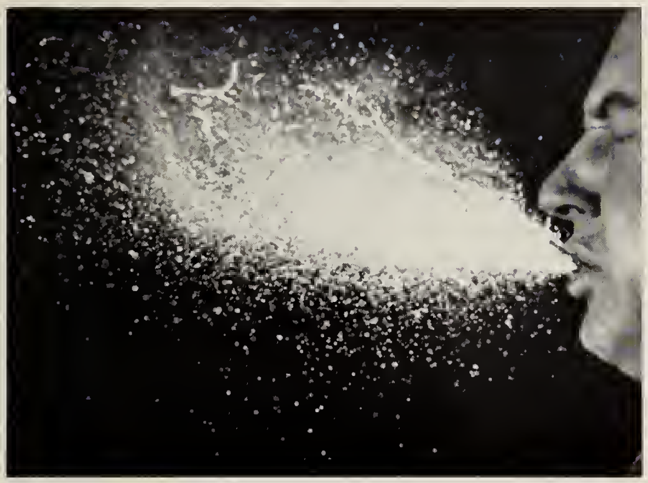
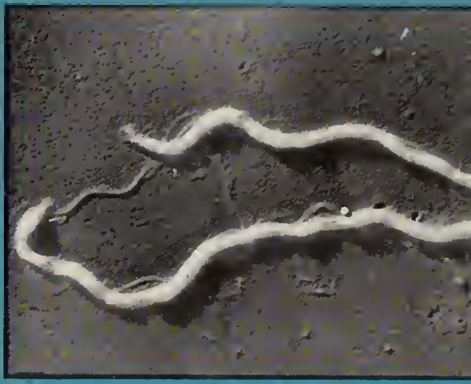
Endamoeba gingivalis in mouth smear, carbol fuchsin and methylene blue. Magnification, Ca. 950X.

**Fig. 4**



A subject with a bad cold, electric spark photograph of a sneeze. Note that the bacteria laden material leaves from between the lips and most of it originates from the oral mucosa. (From Jennison, Scientific Monthly, Jan. 1941)

**Fig. 6**



Treponema pallidum 7200X, enlarged to 17,000X. Electron photomicrograph. (Courtesy E. S. Hampp, D.D.S., A.D.A. Research Associate at the National Institute of Dental Research)

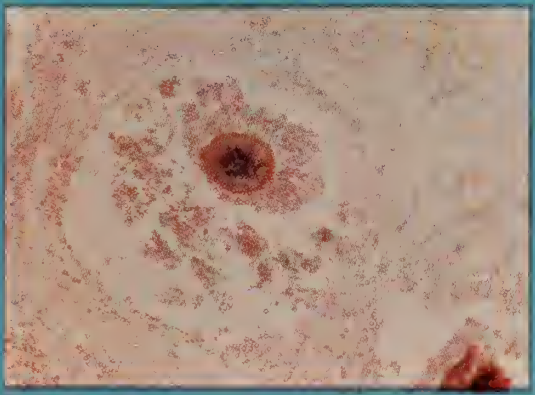


**Fig. 5**

Actinomyces bovis in tissue obtained for biopsy, hematoxylin and eosin. Magnification, ca. 450X.

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PUBLICATIONS: Contributions: Fields of Otolaryngology, Speech and Hearing Rehabilitation and Postgraduate Education. Chief Associate Editor (Otolaryngology), Cyclopedia of Medicine and Surgical Specialties.

HONORS: Sigma Xi, Alpha Omega Alpha, Diplomate American Board of Otolaryngology, Fellow American College of Surgeons.

## OTORHINOLOGICAL ASPECTS

Otorhinology is closely allied to modern dentistry. Overlapping dimensions, a similarity of symptoms and signs common to many entities require careful and complete inventory. The earache, the headache and the facial neuralgias with their dento-sinal connotations, are but a few of the symptoms which challenge the efforts of both the dentist and otolaryngologist. There is ample evidence that this coordinated therapeutic relationship offers improved diagnostic and prognostic possibilities for the patient.

We have made progress in the newer knowledge of chemotherapy, antibiotics, in the evaluation of operative risks and in the stabilization of more precise principles of surgical technics and management of the patient. This forcefully reminded us that while there may be numerous approaches to the employment of these modalities for treatment, there is only one correct diagnosis. For this realization, a routine is recommended to reduce to a minimum the omission of important data and findings. While the specialties of dentistry and otolaryngology deal with separate fields, the organs or parts involved are neither anatomically nor physiologically or pathologically independent but are integral parts of the individual human economy.

Differentiation of conditions with a similar symptomatology offers a challenging task. Since diagnosis implies the determination of the nature of disease to insure a logical, efficient and effective treatment sequence, emphasis on this score is reliably placed. The study of the total person and the thorough inventory of the patient's physical status will avoid the pitfalls of an erroneous diagnosis, the embarrassment of a fallacious prognosis and the blunder of inappropriate or misdirected medical or surgical management.

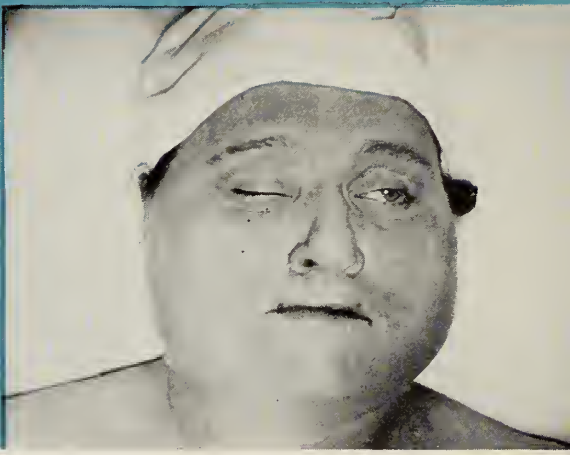
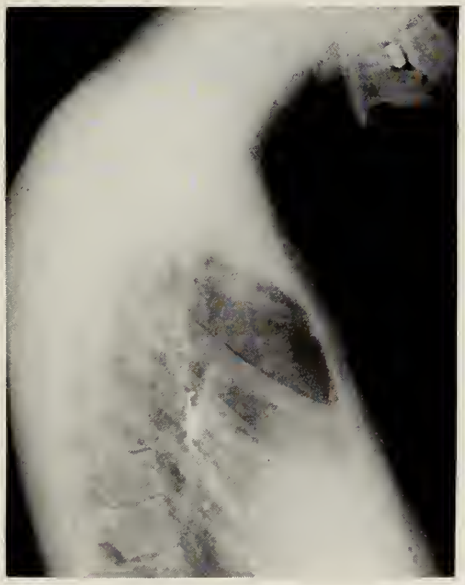


**Fig. 3**

Bilateral suppurative parotitis. There was purulent drainage from the openings of the parotid ducts into the mouth. Note that the left eye is open although the patient is comatose, a result of pressure of the parotid swelling upon the facial nerve. (Courtesy, Sarnat, Bernard G.)

The maxillary sinus is of particular interest to our respective specialties. We should be aware of the fact however that all sinuses may become affected in inflammatory processes through continuity and contiguity of structure, foramina and dehiscences and vascular channels. This sinus is of interest in operations on the teeth of this region and the reverse is also true as sinus surgery may affect the teeth. Furthermore, the region of the maxillary sinus and floor of the nose is a favorite site for various types of cysts and also for benign and malignant neoplasms which arise both within and external to the sinus cavity.

Of prime consideration, too, is the fact that the connective tissue spaces of the pharynx and neck may be the seat of suppuration from infections of dental origin. Other sources of suppuration may be infected tonsils, mastoid disease, retropharyngeal adenitis, parotitis, pharyngeal trauma or post-surgical complications.



**FRANCIS L. LEDERER,**
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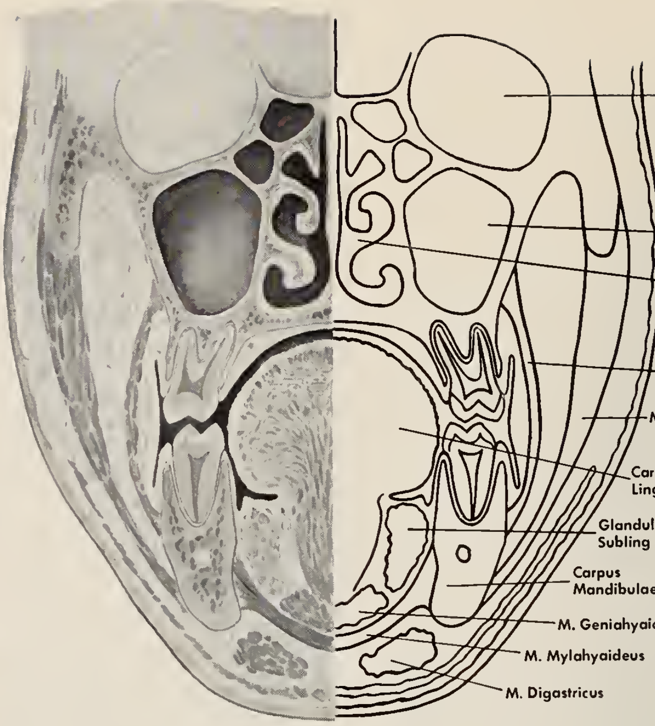

# OTORHINOLOGICAL ASPECT

**Fig. 1**

Frontal section through the head in the region of the first molar. Note the relationship of the maxillary sinus to the roots of the molar, the palate and the nasal cavity. (Courtesy, Massler, Maury and Schour, Isaac: Atlas of the Mouth, American Dental Association, Chicago, 1948.)

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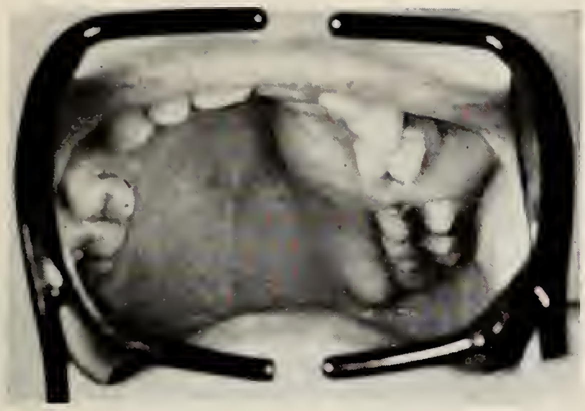
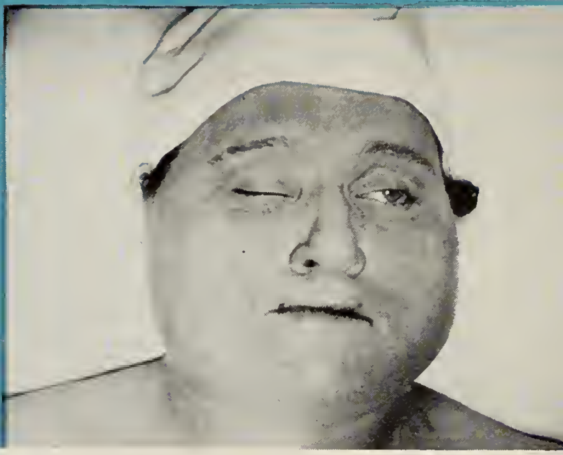


**Fig. 2**

Osteogenic sarcoma of the maxillary sinus. The first evidence of this tumor was its encroachment upon the oral cavity. Note swelling, both lingually and buccally on the left side of the maxilla.

**Fig. 3**

Bilateral suppurative parotitis. There was purulent drainage from the openings of the parotid ducts into the mouth. Note that the left eye is open although the patient is comatose, a result of pressure of the parotid swelling upon the facial nerve. (Courtesy, Sarnat, Bernard G.)



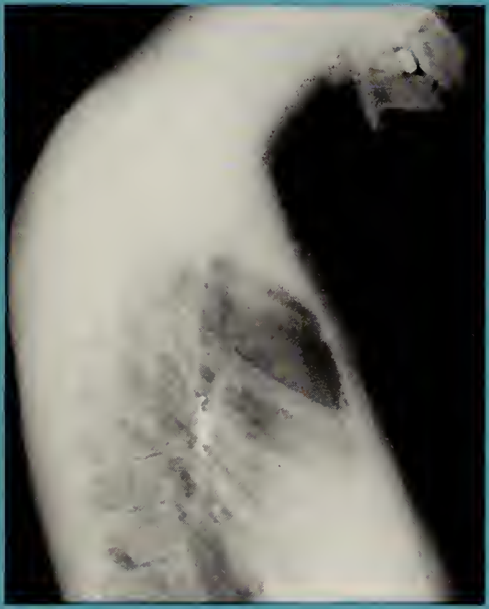


**Fig. 4**

Lateral roentgenogram of the chest of a patient showing a removable dental prosthesis lodged in the right main bronchus. This foreign body was successfully removed by means of bronchoscopy.

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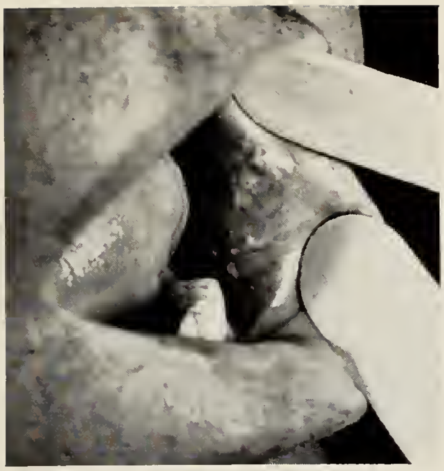
HONORS: Sigma Xi, Alpha Omega Alpha, Pi Kappa Epsilon, President, American Board of Dermatology and Syphilology, Fellow American College of Physicians.

## DERMATOLOGIC ASPECTS

The tongue, buccal mucosa, and the lips are the sites of a wide variety of disorders. These many conditions do not fall in the domain of any single special field of medicine or dentistry. Consequently, in this group of disorders we find much of interest for the dentist, the oral surgeon, the internist, and the dermatologist. Some of these diseases are accepted as properly belonging in one single field; but with others, two or more of the specialties may find something of interest. As a result, it may be said that only exceptionally does one meet with a practitioner who is conversant with all of the diseases affecting these parts.

In some instances, the pathological process is a purely local one; in others, the changes may be due to some systemic disorder; while in still others, the involvement of the mucosa may be only a part of some disorder affecting the skin primarily. Such lesions of the mucous membranes usually present some modifications making their characteristics somewhat different from that seen in the corresponding lesions upon the skin. It is also possible that certain lesions belonging to this last category may appear upon the mucous membranes without the usual accompanying lesions upon the skin. Since some of these conditions are of an infectious nature, while others are not, and since some of them may lead to serious interference with the general health, while others are entirely benign, it is important that the dentist should have some familiarity with at least the more common of these conditions.

Erosive lesions corresponding to vesicular or bullous lesions upon the skin are produced by several conditions, notably by the ingestion of drugs and in pemphigus, erythema multiforme, and dermatitis herepetiformis. Since the prognosis is very bad in pemphigus, while good in the other



conditions, it is important that one keeps all of these conditions in mind.

The mucous membranes reflect particularly the effects of several types of avitaminosis and these sites may give us the first clue as to the background of systemic complaints due to the same lack of vitamins.

Cancer, syphilis, and tuberculosis are among the most serious diseases affecting the mucous membranes, and not infrequently great judgment is required in establishing the differential diagnosis between them.

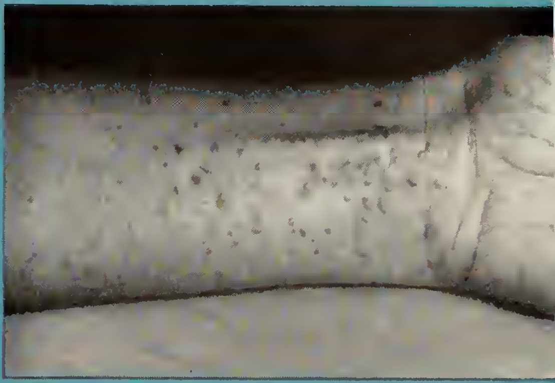


**Fig. 1b**

**Lichen planus of the wrist.** If the mucous membrane involvement is accompanied by lesions on the skin, these are most apt to be seen on the flexor aspect of the wrist and upon the external genitalia, they are characterized by an angular shape of the lesions which are flat-topped, often with central umbilication. The surface of the lesion is frequently traversed by fine, whitish striae. The lesions often occur in a linear or annular arrangement.

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## DERMATOLOGIC ASPECTS

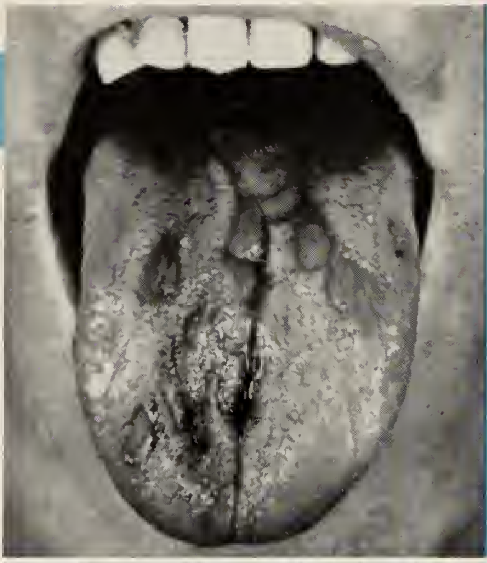

**Fig. 2a**
**Syphilis of the tongue.** In the secondary period, the mucous patches are the characteristic lesion. These are erosive papules covered with a dirty, whitish membrane.

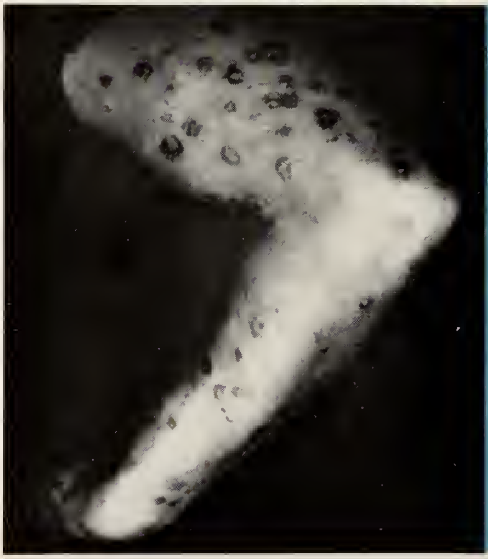

**Fig. 2b**
**Syphilitic eruption of the skin of the arm.** The eruptive manifestations of the secondary period of syphilis are macules or papules. The latter type is discrete, indurated, asymptomatic lesions of dull red to reddish-brown color.

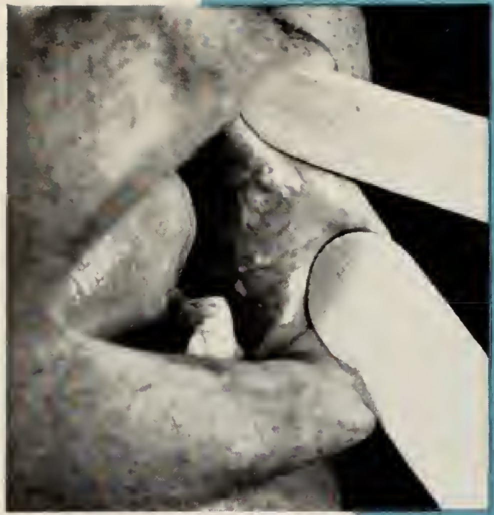

**Fig. 1a**

**Lichen planus of the buccal mucosa.** Lichen planus of the mouth frequently involves the buccal mucosa, especially in the area opposite the molars. Clinically, this is characterized by the presence of whitish lines or circles with a mosaic arrangement in the mucous membranes. The lesions are asymptomatic. The mucosal lesions are frequently mistakenly diagnosed as leukoplakia.

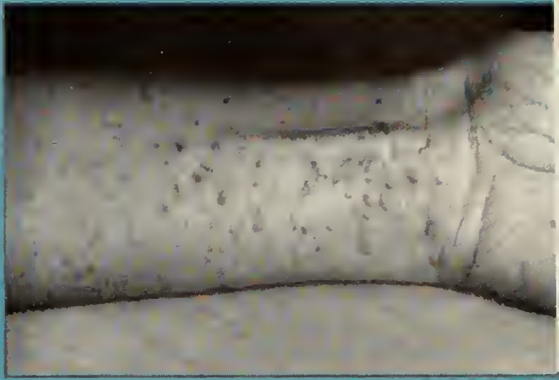

**Fig. 1b**

**Lichen planus of the wrist.** If the mucous membrane involvement is accompanied by lesions on the skin, these are most apt to be seen on the flexor aspect of the wrist and upon the external genitalia, they are characterized by an angular shape of the lesions which are flat-topped, often with central umbilication. The surface of the lesion is frequently traversed by fine, whitish striae. The lesions often occur in a linear or annular arrangement.

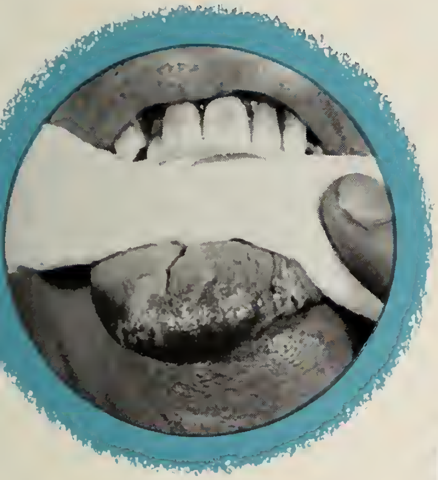

**Fig. 3a**

Moniliasis of the tongue. Moniliasis often involves the mucous membranes and frequently in association with monilial infection of other parts of the body. In adults, lesions of the mucous membranes are membranous in type and frequently show rather bizarre arrangement as in the case illustrated here.

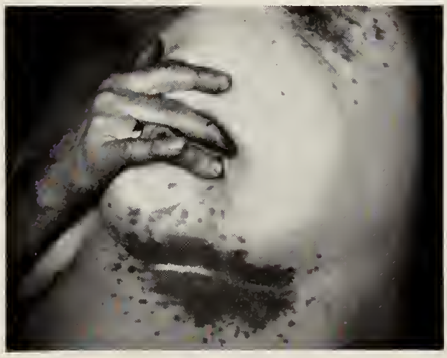

**Fig. 3b**

**Moniliasis of the skin.** Accompanying this is a typical picture of infection with the yeast-like organisms involving the inframammary region and the axilla.

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PUBLICATIONS: Researches in iron metabolism, pathogenesis of anemias, physiology of red blood cell formation and therapeutic use of radioactive phosphorus.

HONORS: Editorial boards of *Blood* and the *Journal of Laboratory and Clinical Medicine* (Editor 1944-49) American Association of Physicians, American Society for Clinical Investigation.

## HEMATOLOGIC ASPECTS

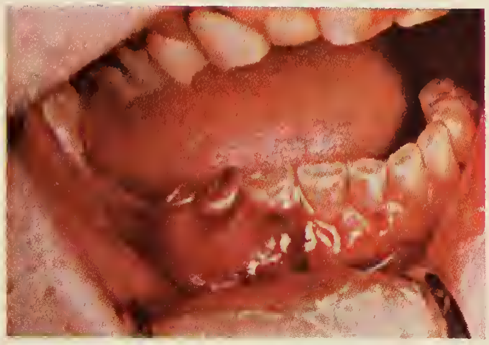
During the past twenty-five years, hematology has become one of the major branches of clinical medicine. Exciting discoveries such as the value of liver and vitamin B in the treatment of patients with pernicious anemia, the importance of vitamin K and prothrombin in blood coagulation, and the significance of the Rh factor have focused attention on the blood dyscrasias. At the same time, improved methods of laboratory diagnosis have demonstrated that these diseases can no longer be regarded as rare. Anemias are common the world over. Leukemias and the malignant diseases of lymph nodes are not only recognized more accurately, but also seem to be increasing in frequency. Bleeding abnormalities complicate the lives of many people.

These considerations are important to dentistry because blood diseases are frequently accompanied by lesions of the oral cavity. Ulcerations, hemorrhage, or infiltration of tissues may be the first clinical manifestations of the disease, and may provide the first hint of the diagnosis. Their correct management requires both precise diagnosis and close cooperation between dentist and physician. Recent advances in the treatment of leukemias and malignant lymphomas have accentuated rather than lessened this need. It is important, for instance, to recognize that nitrogen mustard therapy causes maximum depression of white blood cells and of platelets about fourteen to twenty-one days after it is given. The dangers of hemorrhage and of infection are maximal at that time. Anti-folic acid compounds, currently being evaluated for their effectiveness in acute leukemias, may cause ulcerations indistinguishable from those produced by the leukemic process itself. The course of leukemia may be accelerated by dental surgery, but if the leukemia is first treated and the patient protected from infection with antibiotics, troublesome dental problems may frequently be cared for without trouble.

Significant advances have also been made in the treatment of hemorrhagic disorders, but here again it is important to define the abnormality. It is futile to give vitamin K if the prothrombin is normal, dramatically effective if the prothrombin is low. Calcium apparently is never of value as a hemostatic agent. Local use of thrombin plus transfusions of whole blood or of the antihemophilic fraction of plasma will often control bleeding in patients with hemophilia, or make necessary surgery possible. If hemorrhage occurs because platelets are deficient, the therapeutic approach must be entirely different. In some instances, splenectomy may be required.

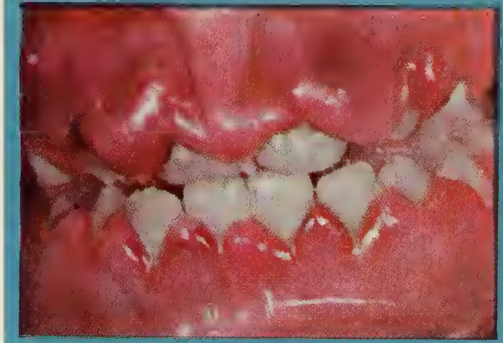
In the management of blood diseases, dentistry and medicine are certainly interdependent. With intelligent management, however, patients with these diseases can frequently be given extra months of useful, comfortable life, even though cures in many instances are still not possible.

**Fig. 2**



Dusky deep red color of mucous membrane with hemorrhagic tendency in a 59 year old white male. RBC found to be 8.5 million per mm$^3$.

**Fig. 4**



Hyperplasia of the gingivae in an adult female, (40 years old) with acute myeloblastic anemia. The initial complaint was ulceration and bleeding of the gingivae. Sternal puncture showed an enormous increase in myeloblastic cells and many mitotic figures.

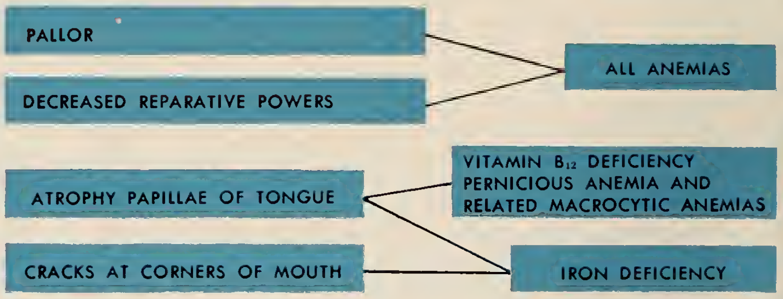
# BLOOD DISEASES

**CARL V. MOORE,**
**A.B., M.D.**

## HEMATOLOGIC ASPECTS

## RED BLOOD CELL DISEASES

### Anemias



PALLOR

DECREASED REPARATIVE POWERS

ALL ANEMIAS

ATROPHY PAPILLAE OF TONGUE

CRACKS AT CORNERS OF MOUTH

VITAMIN $B_{12}$ DEFICIENCY PERNICIOUS ANEMIA AND RELATED MACROCYTIC ANEMIAS

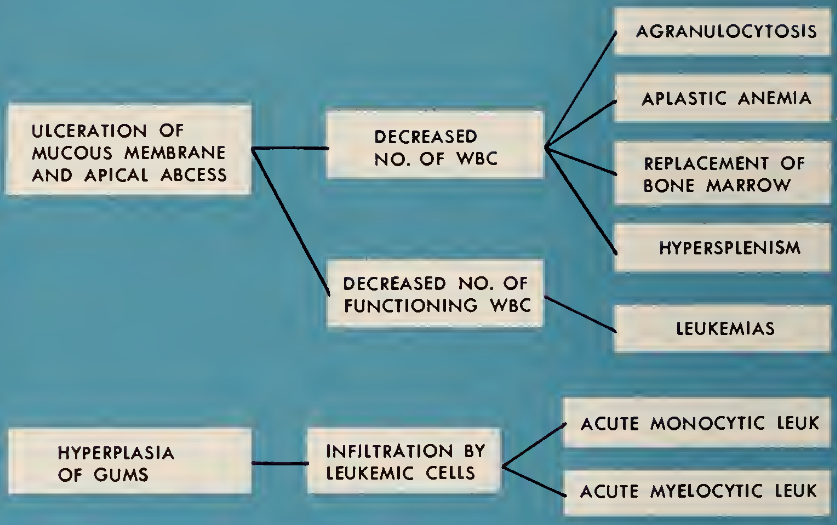
IRON DEFICIENCY

### Polycythemia

DUSKY DEEP RED COLOR OF MUCOUS MEMBRANES

HEMORRHAGIC TENDENCY — CAUSE OFTEN UNKNOWN

## WHITE BLOOD CELL DISEASES



ULCERATION OF MUCOUS MEMBRANE AND APICAL ABCESS

DECREASED NO. OF WBC

AGRANULOCYTOSIS

APLASTIC ANEMIA

REPLACEMENT OF BONE MARROW

HYPERSPLENISM

DECREASED NO. OF FUNCTIONING WBC

LEUKEMIAS

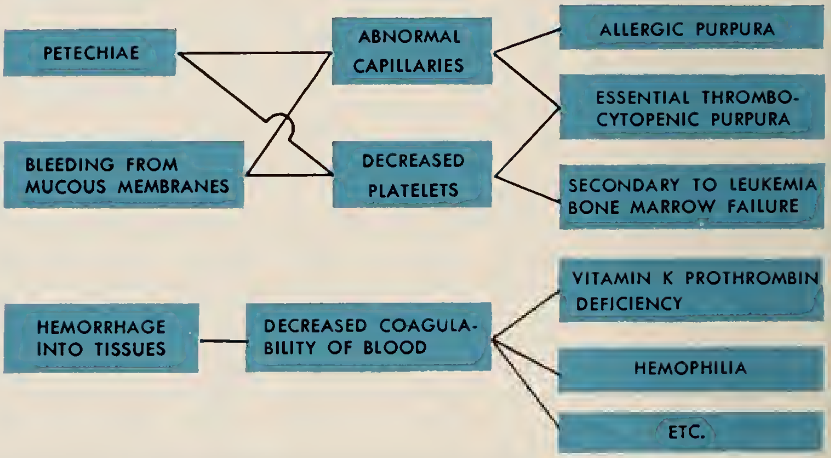
HYPERPLASIA OF GUMS

INFILTRATION BY LEUKEMIC CELLS

ACUTE MONOCYTIC LEUK

ACUTE MYELOCYTIC LEUK

## HEMORRHAGIC DISEASES



PETECHIAE

BLEEDING FROM MUCOUS MEMBRANES

ABNORMAL CAPILLARIES

DECREASED PLATELETS

ALLERGIC PURPURA

ESSENTIAL THROMBOCYTOPENIC PURPURA

SECONDARY TO LEUKEMIA BONE MARROW FAILURE

HEMORRHAGE INTO TISSUES

DECREASED COAGULABILITY OF BLOOD

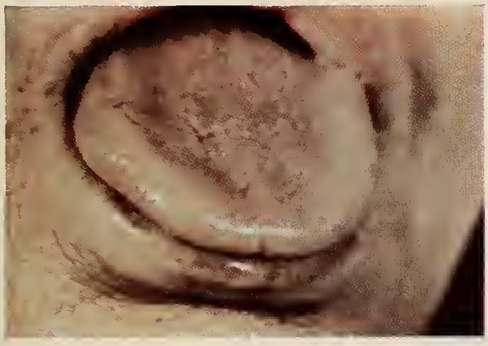
VITAMIN K PROTHROMBIN DEFICIENCY

HEMOPHILIA

ETC.

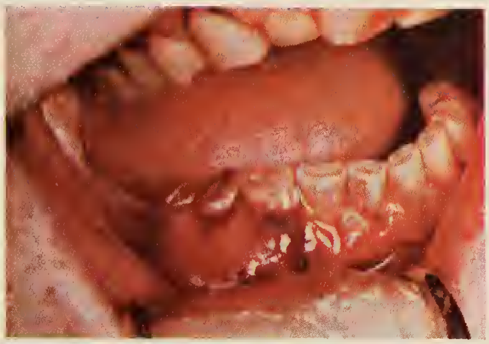
The above table is by no means complete; it serves only to emphasize the most common oral manifestations of the blood diseases. In certain dyscrasias, i.e. aplastic anemia and the leukemias, all three types of formed elements are involved. The clinical manifestations, therefore, may be any combination of those related more specifically to abnormalities of red cells, white cells, or platelets.

**Fig. 1**



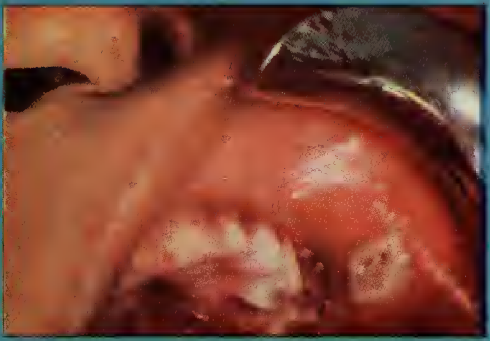
Pallor and atrophy of papilla of tongue in a patient with aplastic anemia. This white female, age 60 years, complained of weakness, dizziness, precordial pain and short windedness.

**Fig. 2**



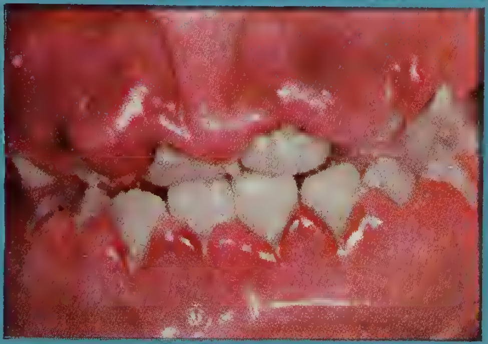
Dusky deep red color of mucous membrane with hemorrhagic tendency in a 59 year old white male. RBC found to be 8.5 million per $mm^3$.

**Fig. 3**



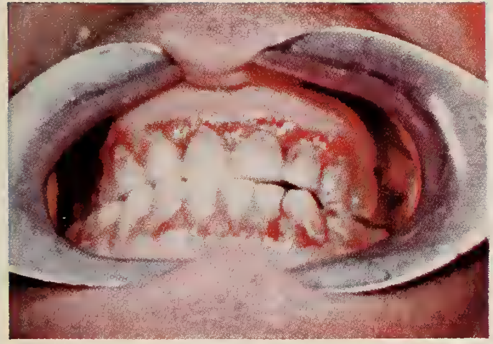
Ulceration of mucous membrane of cheek in a 6 year old Negro girl with acute leukemia.

**Fig. 4**



Hyperplasia of the gingivae in an adult female, (40 years old) with acute myeloblastic anemia. The initial complaint was ulceration and bleeding of the gingivae. Sternal puncture showed an enormous increase in myeloblastic cells and many mitotic figures.

**Fig. 5**



Bleeding of gingiva in a patient with purpura hemorraghica. Initial diagnosis of scurvey was found to be incorrect after blood analysis.

# NOTES

## DISEASES OF THE MOUTH

# QUESTIONS

## NOTES

## LESIONS LOCAL IN ORIGIN

### DENTAL ASPECTS

1. What are some of the substances which produce stomatitis venenata?

2. What is the most frequent cause of red, swollen tissues under dentures?

3. Does burning tongue occur without any demonstrable organic changes?

4. What are the three lesions of the oral mucosa which are not tumors but which most frequently have led to cancerphobia in your experience?

5. How frequently is torus palatinus encountered and from what must it be differentiated?

### BACTERIOLOGIC ASPECTS

6. What is the relative importance of the oral cavity as a reservoir of air-borne infection?

7. What is the relation of oral bacteria to postoperative parotitis, postoperative pneumonia, secondary infection of tuberculous lesions of lungs?

8. What are the effects of saliva on mouth bacteria?

9. What effect have troches or chewing gum, containing antibacterial substance (e.g. sulfonamides, antibiotics) on the oral bacteria?

10. What is Vincent's disease? Is it transmissible?

### OTORHINOLOGICAL ASPECTS

11. A patient presents himself with the complaint of pain in some of the upper teeth. Examination of the teeth does not reveal any accountable pathologic findings. What could be the cause and what steps should be taken to determine the cause?

## NOTES

## QUESTIONS

12. What are the underlying factors in the creation of an antra-oral fistula and how should this be managed?

13. What structures should be examined during a routine dental check up?

14. Following dental extractions, a patient develops cough with or without fever. State the possible cause and what examinations are imperative.

15. What are some of the causes of enlargement of the submaxillary and cervical lymph nodes?

### LESIONS SYSTEMIC IN ORIGIN

#### DERMATOLOGIC ASPECTS

16. What are the chief mucous membrane changes resulting from avitaminosis?

17. Are drugs taken systemically or locally apt to produce lesions of the mucous membranes?

18. What types of virus infections affect the mucous membranes?

19. What disorders give rise to whitish lesions of the mucous membranes?

20. Is leukoplakia always a dangerous lesion from the standpoint of malignant change?

#### HEMATOLOGIC ASPECTS

21. When would you order blood studies on a patient?

22. Is there any relationship between successful denture *therapy* and systemic evaluation of the patient suspected of pernicious anemia?

23. What are the main effects of oral infection upon the blood picture?

24. What is the relation of drugs of the amidopyrine group and agranulocytosis?

25. How would you treat a known hemophilic patient requiring an extraction?

# NOTES

# THESE ARE THE REGISTRANTS

| STATE—DENTAL SOCIETY | CITY | LEADER |
|---|---|---|
| **ALABAMA** | | |
| Dothan Dental Society | Dothan | Dr. Dupree Davis |
| Northeast Alabama Dental Society | Gadsden | Dr. Ansley G. Brown |
| Second District Dental Society | Montgomery | Dr. Fred M. Haston |
| Sixth District Dental Society | Tuscaloosa | Dr. Dudley L. Davis |
| **ARIZONA** | | |
| Southern Arizona Dental Society | Tucson | Dr. R. L. Felix |
| **ARKANSAS** | | |
| Fort Smith Dental Society | Fort Smith | Dr. I. M. Sternberg |
| Central District Dental Society | Little Rock | Dr. E. D. Jernigan |
| Ozark Dental Study Club | Rogers | Dr. Robert G. Pickard |
| **CALIFORNIA** | | |
| Kern County Dental Society | Bakersfield | Dr. Lyford Pruitt |
| Fifth District Dental Society | Fresno | Dr. Julius Yee |
| Third District Dental Society | Long Beach | Dr. Arthur C. Meigs |
| Los Angeles Study Group | Los Angeles | Dr. Bernard Z. Rabinowitch |
| Alameda County District Dental Society | Oakland | Dr. J. Elliott Dunn |
| Second District Dental Society | Pasadena | Dr. H. R. Benedict |
| Riverside Dental Association | Riverside | Dr. W. E. Jenkins |
| San Diego County Dental Society | San Diego | Dr. C. B. Reid |
| San Francisco Dental Society | San Francisco | Dr. E. J. Lattig |
| Santa Clara District Dental Society | San Jose | Dr. R. T. Dunkin |
| San Mateo County Dental Society | San Mateo | Dr. Victor J. Matheu |
| Marin County Dental Society | San Rafael | Dr. Jack Gazzola |
| Sixteenth District Dental Society | Santa Maria | Dr. L. A. Pereira |
| Santa Barbara-Ventura County Dental Society | Ventura | Dr. Douglas E. Burns |
| Ninth District Dental Society | Watsonville | Dr. Oscar Nusz |
| **COLORADO** | | |
| Colorado Springs Dental Society | Colorado Springs | Dr. Robert E. Ehlert |
| Denver Dental Association | Denver | Dr. W. E. Mulford |
| **CONNECTICUT** | | |
| Hartford Dental Society | Hartford | Dr. Joseph Nassau |
| New Britain Dental Society | New Britain | Dr. Joseph M. Lobo |

## DELAWARE

| | | |
|---|---|---|
| Delaware State Dental Society | Wilmington | Dr. Martin B. Yalisave |

## DISTRICT OF COLUMBIA

| | | |
|---|---|---|
| Howard University College of Dentistry | Washington | Dr. Russell A. Dixon, Dean |

## FLORIDA

| | | |
|---|---|---|
| Broward County Dental Society | Fort Lauderdale | Dr. Castles W. Moore |
| Jacksonville Dental Society | Jacksonville | Dr. Dudley L. Reep |
| Orange County Dental Society | Orlando | Dr. George C. Shuman |
| Bay County Dental Society | Panama City | Dr. J. L. Hester |
| Pensacola Dental Society | Pensacola | Dr. William H. Caker |
| Pinellas County Dental Society | St. Petersburg | Dr. Cameron Forbes |
| Hillsborough County Dental Society | Tampa | Dr. Leon Schwartz |

## GEORGIA

| | | |
|---|---|---|
| Fifth District Dental Society | Atlanta | Dr. Marvin Goldstein |

## ILLINOIS

| | | |
|---|---|---|
| St. Clair District Dental Society | Belleville | Dr. T. J. Winkler |
| Southern Illinois District Dental Society | Benton | Dr. W. A. McKee |
| McLean County Dental Society | Bloomington | Dr. R. A. Chrismon |
| Arcolian Dental Arts Society Study Club | Chicago | Dr. James DeBiase |
| North Town Telephone Extension Forum | Chicago | Dr. Jules Hazelkorn |
| Englewood Branch Chicago Dental Society | Chicago | Dr. Edmund M. Glavin |
| Vermilion County Dental Society | Danville | Dr. D. L. Carpenter |
| Decatur District Dental Society | Decatur | Dr. Thomas J. Campbell |
| Fox River Valley Dental Society | Elgin | Dr. Ralph W. Muchow |
| North Suburban Branch Chicago Dental Society | Evanston | Dr. Paul Boss |
| Northwest District Dental Society | Freeport | Dr. George B. Vagelei |
| Madison District Dental Society | Granite City | Dr. Lester Borton |
| U. S. Naval Training Center Dental Department | Great Lakes | Captain C. W. Schantz |
| Morgan County Dental Society | Jacksonville | Dr. Robert W. Herr |
| Will-Grundy Dental Society | Joliet | Dr. Harry B. Hill |
| LaSalle County Dental Society | LaSalle | Dr. Edward J. Shaheen |
| Eastern Illinois Dental Society | Mattoon | Dr. T. E. McMeekon |
| West Suburban Branch Chicago Dental Society | Oak Park | Dr. C. A. Hansan |
| Pike County Dental Society | Pittsfield | Dr. Frederick Berry |
| Winnebago County Dental Society | Rockford | Dr. R. D. Stitzel |
| Rock Island District Dental Society | Rock Island | Dr. Richard P. Hainline |
| Springfield Dental Study Club | Springfield | Dr. J. E. Ketterer |
| Whiteside-Lee Dental Society | Sterling | Dr. W. E. Stern |

## INDIANA

| | | |
|---|---|---|
| Madison County Dental Society | Anderson | Dr. Paul T. Worster |
| First District Dental Society | Evansville | Dr. M. D. Bronson |
| Isaac Knapp Dental Study Club | Fort Wayne | Dr. Marion J. Calbeck |
| Northwest Indiana Dental Society | Gary | Dr. Wayne K. Stoler |
| Indianapolis Dental Society | Indianapolis | Dr. D. C. Barnhill |
| West Central Dental Society | LaFayette | Dr. Paul A. Risk |
| Wabash Valley Dental Society | Marion | Dr. George Richardson |
| Delaware County Dental Society | Muncie | Dr. Larry Long |
| Richmond Dental Association | Richmond | Dr. Walter A. Crum |
| Western Indiana Dental Society | Terre Haute | Dr. William Aitken |

## IOWA

| | | |
|---|---|---|
| Des Moines County Dental Society | Burlington | Dr. K. N. Kruse |
| Clinton County Dental Society | Clinton | Dr. M. C. Hurley |
| Council Bluffs Study Club | Council Bluffs | Dr. Eugene L. Marsh |
| Scott County Dental Society | Davenport | Dr. Robert J. Getman |
| DesMoines District Dental Society | Des Moines | Dr. J. P. Pinkerton |
| Dubuque County Dental Society | Dubuque | Dr. A. J. Kolb |
| Fort Dodge District Dental Society | Fort Dodge | Dr. A. M. Oosterhuis |
| Southwestern Iowa District Dental Society | Red Oak | Dr. Harry R. Carney |
| Sioux City Dental Society | Sioux City | Dr. Elmer A. Rizk |
| Waterloo Dental Society | Waterloo | Dr. M. E. Moule |

## KANSAS

| | | |
|---|---|---|
| Hutchinson Dental Study Club | Hutchinson | Dr. R. M. White |
| Geary County Dental Society | Junction City | Dr. L. W. Stewart |
| Shawnee County Dental Society | Topeka | Dr. J. I. Cosper, Jr. |

## KENTUCKY

| | | |
|---|---|---|
| West Central Dental Society | Dawson Springs | Dr. J. W. Jones, Jr. |
| Blue Grass Dental Society | Lexington | Dr. George Prewitt |
| Louisville District Dental Society | Louisville | Dr. J. G. Mixson |
| Southwestern Kentucky Dental Society | Paducah | Dr. E. G. Stamper, Jr. |

## LOUISIANA

| | | |
|---|---|---|
| Sixth District Dental Society | Baton Rouge | Dr. James C. Brousseau |
| Lake Charles Dental Study Club | Lake Charles | Dr. John D. Lewis |
| Third District Dental Society | New Iberia | Dr. J. W. Emmer |
| New Orleans Dental Association | New Orleans | Dr. Wilbur Lazarus |

## MAINE

| | | |
|---|---|---|
| Androscoggin Dental Society | Lewiston | Dr. William E. Fahey |
| Greater Portland Dental Society | Portland | Dr. Arthur Pitzele |

## MARYLAND

| | | |
|---|---|---|
| Baltimore Study Club | Baltimore | Dr. B. R. Pollack |
| Allegany-Garrett County Dental Society | Cumberland | Dr. J. Russell Cook |

## MASSACHUSETTS

| | | |
|---|---|---|
| Holyoke Dental Society | Holyoke | Dr. R. E. Goddu |
| Massachusetts Dental Society | Lawrence | Dr. M. F. Grossman |
| Chapter of World War II Veterans | Pittsfield | Dr. S. K. Gregory |
| North Shore Dental Study Club | Salem | Dr. Warren R. Mayne |
| Valley District Dental Society | Springfield | Dr. Arthur Gold |
| Worcester Dental Society | Worcester | Dr. Philip M. Kramer |

## MICHIGAN

| | | |
|---|---|---|
| Battle Creek Dental Society | Battle Creek | Dr. Donald Whitman |
| Kalamazoo Dental Society | Kalamazoo | Dr. H. W. Fountain |
| Thumb District Dental Society | Port Huron | Dr. C. W. Carter |
| Saginaw County Dental Society | Saginaw | Dr. J. W. Boell |

## MINNESOTA

| | | |
|---|---|---|
| Duluth District Dental Society | Duluth | Dr. Arthur G. Peterson |
| Range Dental Study Club | Hibbing | Dr. J. E. Hoffman |
| Minneapolis District Dental Society | Hutchinson | Dr. L. M. Ingebrigtsen |
| Minneapolis District Dental Society | Minneapolis | Dr. L. M. Ingebrigtsen |
| Rochester Dental Society | Rochester | Dr. R. H. Oleson |
| Winona County Dental Society | Winona | Dr. L. L. Korda |

## MISSISSIPPI

| | | |
|---|---|---|
| Northwest Mississippi Dental Society | Greenwood | Dr. A. H. Richter, Jr. |
| East Mississippi Dental Society | Hattiesburg | Dr. C. S. Williams, Jr. |

## MISSOURI

| | | |
|---|---|---|
| Columbia Dental Society | Columbia | Dr. John M. Kingsley |
| Southwest District Dental Society | Joplin | Dr. J. S. Ritchey |
| St. Louis County Dental Society | St. Louis | Dr. Randal Culver |
| Springfield District Dental Society | Springfield | Dr. Frank L. Canedy |
| Northwest Missouri District Dental Society | St. Joseph | Dr. Isaac H. Droher |

## MONTANA

| | | |
|---|---|---|
| Yellowstone Dental Society | Billings | Dr. E. S. Weyer |
| Third District Dental Society | Butte | Dr. E. J. Guay |
| Second District Dental Society | Missoula | Dr. T. T. Rider |

## NEBRASKA

| | | |
|---|---|---|
| Hall County Dental Society | Grand Island | Dr. W. H. Luers |

| | | |
|---|---|---|
| Adams County Dental Society | Hastings | Dr. Herbert Seberg |
| Lincoln County Dental Society | Lincoln | Dr. D. T. Waggener |
| North Nebraska Dental Association | Norfolk | Dr. A. P. Herbst |
| Omaha District Dental Society | Omaha | Dr. Arlo M. Dunn |

## NEW HAMPSHIRE

| | | |
|---|---|---|
| New Hampshire Dental Society | Manchester | Dr. Robinson C. Frost |

## NEW JERSEY

| | | |
|---|---|---|
| Monmouth County Dental Society | Asbury Park | Dr. Nathan Epstein |
| Southern Dental Society of New Jersey | Camden | Dr. Luther M. Mkitarian |
| Hudson County Dental Society | Jersey City | Dr. J. E. Grodjesk |
| Tri-County Dental Society | Morristown | Dr. C. K. Botkin |
| Passaic County Dental Society | Passaic | Dr. R. Feltman |
| Plainfield Dental Society | Plainfield | Dr. Clifford W. Doeringer |
| Mercer Dental Society | Trenton | Dr. James R. Burns |

## NEW MEXICO

| | | |
|---|---|---|
| Albuquerque District Dental Society | Albuquerque | Dr. William A. Blueher |
| Deming Dental Society | Deming | Dr. Judson J. Calhoun |

## NEW YORK

| | | |
|---|---|---|
| Broome County Dental Society | Binghamton | Dr. Robert S. Brooks |
| Brownsville and East New York Dental Society | Brooklyn | Dr. M. B. Rubin |
| Buffalo Dental Society | Buffalo | Dr. Carlton W. Meyer |
| Chemung County Dental Society | Elmira | Dr. Milton Jacobson |
| Tompkins County Dental Society | Ithaca | Dr. George J. Visnyei |
| Jamestown Dental Society | Jamestown | Dr. B. G. Quackenbush |
| Fulton, Hamilton and Montgomery Dental Society | Johnstown | Dr. J. F. Lindsay |
| Illinois Dental Club | New York | Dr. M. B. Rubin |
| Columbia Study Club | New York | Dr. George Stein |
| Cattaraugus County Dental Society | Olean | Dr. R. L. Burt |
| Upper Hudson Dental Society | | |
| Saratoga County Dental Society | Saratoga Springs | Dr. L. W. Roohan, Jr. |
| The Onondaga Academy of Dentists | Syracuse | Dr. Kenneth W. McMahon |
| Utica Study Group | Utica | Dr. J. F. Folley |
| Jefferson County Dental Society | Watertown | Dr. A. A. Hewett |

## NORTH CAROLINA

| | | |
|---|---|---|
| Buncombe County Dental Society | Asheville | Dr. Dean H. Crawford |
| Charlotte Dental Society | Charlotte | Dr. Thomas G. Nisbet |
| Forsyth County Dental Society | Winston-Salem | Dr. D. A. Jackson |

## NORTH DAKOTA

| | | |
|---|---|---|
| Bismarck Study Group | Bismarck | Dr. F. O. Beck |

## OHIO

| | | |
|---|---|---|
| Muskingum Valley Dental Society | Cambridge | Dr. E. J. Joseph |
| Stark County Dental Society | Cantan | Dr. Glenn F. Hastetler |
| Alpha Omega Fraternity | Cleveland | Dr. Oscar R. Rasen |
| Daytan Dental Society | Daytan | Dr. Joseph Cackerill |
| Narthwestern Ohia Dental Society | Lima | Dr. Dayne W. Brattain |
| Mansfield Dental Society | Mansfield | Dr. H. R. Oberlin |
| Carydan-Palmer Dental Society | Yaungstawn | Dr. David V. Bender |

## OKLAHOMA

| | | |
|---|---|---|
| Enid Dental Study Club | Enid | Dr. Gearge Webber |
| Oklahama County Study Club | Oklahama City | Dr. M. S. Taubman |
| Narthern District Dental Society | Tulsa | Dr. R. C. Mitchell |

## OREGON

| | | |
|---|---|---|
| Klamath County District Dental Society | Klamath Falls | Dr. Albert E. Burns |
| Sauthern Oregan Dental Society | Medfard | Dr. B. L. Lagesan |
| Partland District Dental Society | Partland | Dr. Walt E. Happe |

## PENNSYLVANIA

| | | |
|---|---|---|
| Mantgamery-Bucks Dental Society | Abingtan | Dr. H. Vernan Lapp |
| Allentawn Dental Society | Allentawn | Dr. Allen C. Brader |
| Blair County Dental Society | Altaana | Dr. Fred Miller |
| Bradfard Dental Society | Bradfard | Dr. D. F. Greer |
| Cumberland Valley Dental Society | Chambersburg | Dr. Danald J. Glen |
| Eastan Dental Society | Eastan | Dr. R. G. Skinner |
| Erie County Dental Society | Erie | Dr. F. A. Stewart |
| Harrisburg Dental Society | Harrisburg | Dr. P. F. McCracken |
| Hazletan District Dental Society | Hazletan | Dr. Reynald Hach |
| Lawrence County Dental Society | New Castle | Dr. A. G. Reynalds |
| Harris Dental Society | New Halland | Dr. C. W. Richmand |
| Mantgamery-Bucks Dental Society | Narristawn | Dr. Henry M. Rasenman |
| Philadelphia County Dental Society | Philadelphia | Dr. Harrisan M. Berry, Jr. |
| Odontological Society of Western Pennsylvania | Pittsburgh | Dr. W. Earle Craig |
| Schuylkill Dental Society | Pattsville | Dr. R. M. Warne |
| Reading Dental Society | Reading | Dr. F. H. Haeffer |
| Scrantan Study Club | Scrantan | Dr. Jahn C. Specker |
| Warren County Dental Society | Warren | Dr. William G. DeFarest |
| Sixth District Dental Society | Williamspart | Dr. C. W. Valckmer, Jr. |
| Yark County Dental Society | Yark | Dr. Charles Dietz, Jr. |

## RHODE ISLAND

| | | |
|---|---|---|
| Pravidence District Dental Society | Pravidence | Dr. W. D. MacIntash |

## SOUTH CAROLINA

| | | |
|---|---|---|
| Charleston Dental Society | Charlestan | Dr. W. C. Hudsan |

## SOUTH DAKOTA

| | | |
|---|---|---|
| Fourth District Dental Society | Aberdeen | Dr. Walter H. Margans |
| Black Hills District Dental Society | Lead | Dr. J. F. Silva |
| Second District Dental Society | Sioux Falls | Dr. L. L. Eggers |

## TENNESSEE

| | | |
|---|---|---|
| Second District Dental Society | Knoxville | Dr. Roy R. Chastain |
| Ninth District Dental Society | Memphis | Dr. F. N. Weber |
| Fifth District Dental Society | Nashville | Dr. Richard H. Sullivan |
| Meharry Medical College | Nashville | Dr. W. H. Allen, Dean |

## TEXAS

| | | |
|---|---|---|
| Seventeenth District Dental Society | Abilene | Dr. Seth B. Cox |
| Amarillo Dental Society | Amarillo | Dr. J. S. Rogers |
| Nueces Valley District Dental Society | Carpus Christi | Dr. B. N. Coward |
| Dallas County Dental Society | Dallas | Dr. H. Loren Miller |
| Fart Warth District Dental Society | Fart Worth | Dr. Carl A. Gibbe |
| Galveston District Dental Society | Galvestan | Dr. William L. Glenn, Jr. |
| Rio Grande Valley Dental Society | Harlingen | Dr. J. G. Brittain |
| Houstan District Dental Society | Hauston | Dr. W. L. Stavall |
| Angelina County Dental Society | Lufkin | Dr. Ned Barrett, Jr. |
| Fourth District Dental Society | McKinney | Dr. Charles H. Saunders |
| Permian Basin Dental Society | Odessa | Dr. J. T. McKissick, Jr. |
| San Antonia District Dental Society | San Antania | Dr. K. Thamas |
| Texarkana Dental Society | Texarkana | Dr. Jahn C. Wyrick |
| Wichita Falls District Dental Society | Wichita Falls | Dr. Harvey B. White, Jr. |

## UTAH

| | | |
|---|---|---|
| Second District Dental Society | Ogden | Dr. Haward L. Call |
| Salt Lake City Dental Society | Salt Lake City | Dr. K. L. Dedekind |

## VIRGINIA

| | | |
|---|---|---|
| Lynchburg Dental Society | Lynchburg | Dr. S. C. Maddox, Jr. |
| Peninsula Dental Society | Newport News | Dr. A. G. Orpahidys |
| Virginia Tidewater Dental Association | Norfolk | Dr. Carl R. Pierce |
| Richmand Dental Society | Richmand | Dr. W. C. Henderson |
| Roanoke Dental Society | Roanake | Dr. Moffett H. Bawman |

## WASHINGTON

| | | |
|---|---|---|
| Spakane District Dental Society | Spokane | Dr. J. Willard Newby |
| Tacama District Dental Society | Tacama | Dr. Danald Shaw |
| Walla Walla Valley Dental Society | Walla Walla | Dr. J. O. Young |

## WEST VIRGINIA

| | | |
|---|---|---|
| Kanawha Valley Dental Society | Charleston | Dr. N. H. Baker |
| Harrison County Dental Society | Clarksburg | Dr. J. C. Thompson |
| Blennerhassett Dental Society | Parkersburg | Dr. Frank R. Young |

## WISCONSIN

| | | |
|---|---|---|
| Outagamie County Dental Society | Appleton | Dr. John E. LeFevre |
| Rock County Dental Society | Beloit | Dr. E. J. Kubal |
| Columbia-Dodge-Marquette Dental Society | Columbus | Dr. R. C. Howe |
| Fond du Lac County Dental Society | Fond du Lac | Dr. H. G. Horneck |
| Jefferson County Dental Society | Jefferson | Dr. C. L. Bergmann |
| LaCrosse District Dental Society | LaCrosse | Dr. H. R. Lawler |
| Dane County Dental Society | Madison | Dr. H. R. Dickert |
| Manitowoc County Dental Society | Manitowoc | Dr. A. J. Zimmer |
| Milwaukee County Dental Society | Milwaukee | Dr. A. T. Bassman |
| Winnebago County Dental Society | Oshkosh | Dr. C. G. Miller |
| Racine County Dental Society | Racine | Dr. Walter Gearen |
| Sheboygan County Dental Society | Sheboygan | Dr. D. J. Roblee |
| Marathon County Dental Society | Wausau | Dr. J. H. Seidl |

## CANADA

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|---|---|---|
| Sydney Dental Society | Sydney, Nova Scotia | Dr. S. D. Florian |
| Northwest Society of Dental Medicine | Vancouver British Columbia | Dr. M. J. Walley |
| Calgary Dental Society | Calgary, Alberta | Dr. R. T. Shillington |
| Moose Jaw Dental Society | Moose Jaw, Sask. | Dr. A. F. Muirhead |
| Saskatoon Dental Society | Saskatoon, Sask. | Dr. Frank Bernbaum |
| Prince Albert Dental Society | Prince Albert, Sask. | Dr. N. A. Dust |
| Halifax Dental Society | Halifax, Nova Scotia | Dr. E. W. Barnstead |
| Winnipeg Dental Society | Winnipeg, Manitoba | Dr. Franklin W. Jones |
| Alpha Omega Fraternity Omega Chapter | Toronto, Ontario | Dr. Irving Appleby |

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